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PERSONAL EXPERIENCES IN VASCULAR SURGERY *

A STATISTICAL SYNOPSIS

RUDOLPH MATAS, M.D.

NEW ORLEANS, LA.

HISTORICAL PREFACE.—Since I am circumscribed by the title of this paper to a summary of my personal experience in the surgery of the blood vessels, with special reference to aneurysm, I trust it will not be regarded as inappropriate that I should begin with a statement of the historical and statistical data upon which this experience is based.

Though the surgery of the blood vessels, meaning by this the operations upon the great arteries and veins that require anatomic knowledge and surgical skill for their performance, constituted one of the most notable and distinctive chapters in the history of surgery in Louisiana, it is not until a relatively late period in the state's history—as late as 1825—22 years after the territory now known as Louisiana had become a possession of the United States, that the first records of such operations appear in the annual reports of the Charity Hospital of New Orleans. Prior to this date, the most careful inquiry into the history of the colonial period, through the 104 years of the alternate French and Spanish dominations (1699–1803), fails to disclose any documents or data that would throw light on the status of surgery in the colony, except for the laws that regulated its practice and which, as late as 1770 (during Governor O'Reilly's administration), discriminated between the physician and the surgeon, and made the latter subservient to the former. This distinction was never strictly observed in the colony, except in the case of the so-called "barber-surgeons," and had ceased altogether after the Louisiana purchase by the United States, in 1803. Though it would seem strange and singular that the records of the early military and other well-qualified colonial surgeons, of great experience in the constant warfare of the colonists with their savage foes and their European rivals, should have been so barren of historical commentaries or official reports, it is none the less true that no records of their surgical services and activities are in existence.

In contrast to the lack of information on the surgery of the colonial period, the medical historian finds a great wealth of material in the epidemic and endemic fevers and other pestilential diseases that frequently ravaged the

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population and which, combined with other great calamities (war, famine, floods, hurricanes, fires), often threatened the struggling colony at New Orleans with extinction. These major evils so completely overshadowed the casualties and incidents of surgical practice that no references to the latter are found in the official reports of the colonial governors, or in the later publications of the historians of that period. In the absence of all records, we can well imagine that, whatever the modes of surgical practice adopted by the French colonial surgeons, they were largely, if not exclusively, modeled after the pattern of the schools of France, the mother country. During the Spanish interregnum (1766–1803) medical practice remained essentially French and unchanged, except in the purely official relations. It is, however, a notable fact, worthy of record, that the first book printed in Louisiana (1796) and translated into French for popular distribution, was a medical treatise, written in Spanish, by Dr. Joseph Masdevall,* physician to King Charles III, and to the royal household.

Surgical practice in Louisiana was, therefore, a mere reflex of the surgery in France as it developed during the eighteenth century, and it is only to this source that we can look for information on the status of surgery in Louisiana and in the other French colonies of the period. When we consider that the tourniquet, invented by Morel (1674), and its modifications by Pettit and others, had not come into general use until the first decade of the century, and then used only to control hemorrhage in amputations; that the ligation of arteries was almost exclusively confined to hemostasis in amputations; that hemostasis in bleeding wounds was largely a matter of local vegetable, animal and mineral styptics, among which flourished the "hemostatic plug or 'button'

* "Médicamens et Précis de la Méthode de M. Masdevall, Docteur médecin du Roi d'Espagne Charles IV., Pour guérir Toutes les Maladies, Epidémiques, Putrides, et Malignes, Fièvre de Différents Genres, etc., pour en Préserver. Divisés en Paragraphes et en Numeros Correspondants, à l'Usage des Familles Dépourvues de Médecins. Prix 4 Escalins Broche, chez Louis Duclet, Imprimeur, à la Nouvelle Orleans, 1796. Avec Permission du Gouvernement." The book is dedicated to the "very illustrious" Baron de Carondelet, Governor of Louisiana.

Masdevall stood high in Spain as a sanitarian and epidemiologist, with special experience in the malignant "pestilential and putrid fevers" (typhus, malaria and yellow fever), in the treatment of which he claimed and was credited with great success. The book printed in New Orleans was intended as a popular guide for the direction of the colonists in the Spanish possessions in the treatment of the malignant fevers. It was published and distributed by order of the King. The chief ingredients of his polypharmacal formula (Masdevall's potion or electuary) were powdered cinchona bark and tartar emetic in small doses. His great merit, and probable secret of his success, was his vigorous condemnation of bloodletting in all its forms, at that time the universal practice. He was President of the Spanish Academy of Medicine, Madrid (1799). He never visited Louisiana. Masdevall was born in Figueras, Catalonia, Spain, and graduated from Cervera and Montpellier. The precise date of his birth is unknown, but it was sometime during the decade 1740-1750. He died at Trujillo (Northern Spain) in 1801, while accompanying King Charles IV and the royal family to the frontier in the prelude to the Napoleonic invasion. (For biography and other data see Garcia del Real, Historia de la Medicina en Espana, 1 vol., 404-418, Madrid, 1921; also Jaume Pi-Suner, in La Medicina Catalana, 8, Nos. 47-48, p. 1-2, August-September, 1937.)

of vitriol, or alum" applied as a surface plug to bleeding vessels—and the actual cautery as a supreme resource; that the ligation of the great arteries, in continuity, for the control of hemorrhage and for the cure of aneurysm was not practiced until 1786, when John Hunter, in England, Desault, Anel, Brasdor, Deschamps, in France, contemporaneously advocated and practiced it; that the first knowledge of arteriovenous aneurysms came with William Hunter, in 1761—and that some of the ablest surgeons of Europe (Percival Pott, 1790, *et al.*) preferred amputations to the ligation of arteries for the cure of aneurysm; that the rules for the ligation of wounded arteries which had been formulated by Larrey, in France, and Guthrie, John Hunter and John Bell, as the result of their observations in the Napoleonic campaigns, were not generally known or universally adopted until after 1825–1840—it is evident, in view of the lateness of these advances, that no ligations involving the blood vessels could have been performed (apart from amputations) during the colonial period, or, at best, only at the close of the eighteenth and beginning of the nineteenth centuries.

Furthermore, the conditions that prevailed in New Orleans, the only center of medical activity in the vast territory or "province" of Louisiana, were not at all favorable or conducive to any unusual or daring surgical enterprises. In 1803, when Louisiana was transferred to the United States, the total population of the city was little over 8,000 and, of these, half were slaves or free blacks. There were in all not over 20 regular graduates or qualified medical practitioners. Apart from the Spanish Military Hospital, and the Leper Hospital, there was only one hospital in effective operation, and that was the Charity Hospital of St. Charles (San Carlos), created in 1784 by a philanthropic Spaniard, Don Andres Almonester y Rojas, to replace the first "Hôpital des Pauvres," a small establishment of a dozen beds, which had been founded by the legacy of an humble French sailor, Jean Louis, in 1736, 18 years after the foundation of New Orleans, in 1718. This had been destroyed by a hurricane in 1779, and the Almonester Hospital itself (with 35 beds) was also consumed by the great fire which swept over the city in 1809. The Spanish Military Hospital, the old "King's Hospital" founded by Bienville and enlarged by the Spanish Governor O'Reilly, in 1770, for sick soldiers and government officials, became obsolete soon after the transfer to the United States, in 1803. Whatever records of the operations performed in these hospitals that could have given an insight into the surgical practice of that period were lost. Furthermore, the messages of the French and Spanish governors to their governments referred only to general medical conditions that affected the health of the colony, but never touched upon matters of technical interest to the profession. Again, we may say that any incentives to the publication of their experiences that the colonial practitioners might have had were denied them by the fact that no printing press was in operation in Louisiana until 1764. The first newspaper published in Louisiana was the *Monitèur de la Louisiane*, which was started near the close of the century, in 1794. Again we may say that the chief stimulus to the exchange and discussion of medical experiences

was wanting for lack of medical organization during the colonial régime. The first attempts at a medical society by O'Reilly in his proclamation of 1770, and by LeDuc in 1804, proved abortive and were not renewed until 1817, when the "Société Médicale de la Nouvelle Orléans," which was composed of the French speaking physicians, was organized (first meeting, August 19, 1817); this was followed in less than three years by the "Physico-Medical Society" (incorporated February 16, 1820), representing the English speaking doctors.

The first medical journal published in Louisiana was the Journal de la Société Médicale de la Nouvelle Orléans, which made its first appearance in 1839, as the organ of the French Society of the same name. The next journal was the New Orleans Medical Journal, edited by Fenner and Hester in 1844, which merged the year after (1845) with the New Orleans Medical and Surgical Journal, which has remained, ever since, as the main repository of the medical history of the state.

We see by all this that, even after the memorable battle of New Orleans on January 8, 1815, there were no medical societies or publications in which events of medical and surgical importance could have been discussed or recorded. Not even the battle of New Orleans, with its nearly 2,000 British killed and wounded, has left behind it any history or historian whose observations could have profited the profession by the study of the medical and surgical lessons gathered from that epochal battle. But there was no Larrey or a Guthrie on either the British or American side to extract out of the carnage of battle something useful for the benefit of humanity and for the progress of surgery, as these great military surgeons had done out of their experience in the Napoleonic Wars.

LOUISIANA PURCHASE AND TRANSFER TO THE UNITED STATES: ITS EFFECT ON THE MEDICAL AFFAIRS OF THE TERRITORY

The radical changes in the political and governmental status of the vast territory which followed the acquisition of the French colonial "Province of Louisiana" by the United States, in 1803, and which became more definite and stabilized when Louisiana was admitted as a state of the Union, in 1812, had a profound effect on the affairs and future of the medical profession of the state as upon everything else.

With the removal of all barriers and restrictions to immigration and commerce with the Anglo-American colonies and foreign countries, which had been imposed by the colonial governments for over a century, the population of New Orleans and of the country districts (parishes) rapidly increased and a new era of business activity, economic and agrarian prosperity was inaugurated which benefited all classes. Paradoxic as it may seem, the Creole descendants of the old French colonists, whose fortunes had remained stagnant during the Colonial regime, now became the greatest beneficiaries of the administrative changes instituted by the new American government. With the free navigation of the Mississippi, the river traffic increased enormously, and with a free outlet to the sea, the unrestrained maritime commerce with foreign countries soon made New Orleans one of the busiest and most pros-

perous ports of the United States. Likewise, a steady stream of hardy Anglo-American pioneers from neighboring and border states, especially from Kentucky, Tennessee, Missouri, Arkansas and Mississippi, soon began to flow into the northwest parishes of the newly created state, and established new English-speaking settlements in parts which had been scarcely touched by the original French and Spanish colonists. The population of New Orleans, which was a little more than 8,000 in 1803, rose to 29,000 in 1821, and, in 1830, had grown to 49,826, of which 29,580 were whites and 21,280 were Negroes or free slaves. The present state of Louisiana (originally defined as the territory of Orleans) comprised a total population of 215,739, of whom 89,441 were whites, 16,710 free colored, and 109,588 slaves. Ten years later, in 1840, the population of New Orleans had increased to 102,204, and that of the state as a whole to 352,411, of whom 158,457 were white inhabitants. The white population was then made up of Anglo-Americans, French and Spanish colonists and their Creole (white) descendants, together with a large proportion of Germans, and a good sprinkling of almost every other nation of the globe.

With this rapid increase in population came many enterprising medical practitioners who hailed from the recently established American schools in the northeastern and southern states, who found lucrative practices in the new Anglo-American population. Owing to the practical cessation of French and Spanish immigration, they soon obtained the ascendancy, not only in medical practice, but in the administration of the medical institutions of the state, which, during the colonial period, had been almost exclusively in the hands of graduates of Paris and other French medical schools.

As the population increased, the need of greater facilities for the care of the indigent sick and injured of New Orleans and of the commonwealth became more pressing. As previously stated, the only charity hospital in active operation at the time of the Louisiana purchase, in 1803, was the Almonester Hospital (San Carlos). This hospital continued in the service of the poor until 1809, when it was consumed in the great fire that swept over the city at that time. After a long interval of great hardship to the poor of the community, a third Charity Hospital, this time erected by the state and not by private beneficence, was built on Canal Street in 1815–1816. It had 125 bed-capacity, and continued to be the main agency for the city's charity to the sick poor until 1832, when it was replaced by a new and spacious building of 540 bed-capacity, which the state erected at the site of the present unit on Common Street, now Tulane Avenue.

At the time of its erection, this hospital was the largest and oldest hospital in the South and probably in the United States. Six years after it had been built, 1848–1849, it was overcrowded, and it became necessary to enlarge the original plant by the addition of new buildings to provide adequate accommodation for the constantly increasing number of indigent sick and injured of the rapidly growing population of the city and state. In 1848, it could accommodate 1,000 patients; and had a yearly average of 11,000 to 13,000 admis-

sions. It was then the largest hospital in existence. It stood fair comparison with those of Europe and America. At that time, in Paris, the Hôtel Dieu had a capacity of 810 beds and La Charité 494 beds, and these were the largest medical charities in that world metropolis.

After serving for more than 103 years, the main building and several other old buildings were demolished in 1936 to make place for the present New Charity Hospital, opened July 1, 1940. It is in the course of the evolution and growth during these 103 years that this institution has attained the magnificent proportions that have made it the largest, as well as the oldest and best known public charity in the Southwest. Its history coincides with the evolution and progress of surgery, and its practice reflects the progressive and revolutionary changes that have characterized the transition from the old to the new surgery, in this centennial period.*

* * * * *

It may be safely said that, from its beginning in 1832 to the late seventies, the Charity Hospital stood as an isolated but towering refuge for the indigent sick and injured, not only of Louisiana but of the sparsely settled country bordering on the banks of the lower Mississippi, who could be transported by the river routes and scant railroad facilities of that time, all the way from Cairo, Illinois, to New Orleans. The relatively unsettled and lawless state of the country all along the Mississippi Valley following the accession of Louisiana and the opening of the newly acquired territory in the South and great Southwest, usually provided an abundant clinic of wounds of all sorts, many of these involving the large peripheral blood vessels, including their sequelae, the arterial and arteriovenous aneurysms. It was these that furnished the most interesting material for the display of anatomic knowledge and surgical skill of the surgeons who flourished at the Charity Hospital and who, in the thirties and forties, were beginning to establish a widespread reputation for New Orleans as the center of the greatest medical and surgical activity in the South and Southwest.

The history of the Charity Hospital, as evolved since 1832, in its present and prodigiously developed plant, is practically contemporaneous with the foundation of the first Medical College of Louisiana, which was organized, in 1834, and continued later (1847–1884) as the Medical Department of the University of Louisiana, and, since 1884, as the Medical School of the Tulane University of Louisiana. As a result of the combined activities of its first faculty, who constituted the medical staff of the hospital, and the great and growing resources of the hospital as a school of incomparable facilities for

* According to the last report, which appeared in March, 1940, the New Charity Hospital is equipped with 3,300 beds, 40 operating rooms; a personnel of 400 visiting physicians and surgeons, 171 interns, 105 resident physicians, 400 graduate nurses, 315 student nurses; also technicians and numerous nonmedical workers included in a paid staff of about 2,200. Statistics for the last year, ending July 1, 1939, show a total of 58,899 admissions; 26,575 new cases and 404,996 total visits in the Out-Clinic; 44,957 cases in the accident room; and 23,473 operations performed in the operating rooms.

clinical instruction, the hospital became, not only the fountain source of medical education in the South, but also, in view of the surgical achievements of the renowned surgical teachers who flourished in it, the very cradle and nursery of surgery in Louisiana. In fact, the history of surgery in so far as Louisiana is concerned, really dates back to the foundation of the Charity Hospital in 1832, for whatever achievements in this field of medicine may have been accomplished by previous generations have been lost and remained unrecorded until the first records of this hospital brought them to light and made them available to the medical historian. It is, therefore, in collecting the data of the history of vascular surgery in which we are now especially concerned, that we are compelled to begin with the first records of the Charity Hospital as the chief and only foundation for our research.

We must bear in mind that it was not until 1845 that anesthesia by ether was discovered, and chloroform in 1847, and that it was not until late in the seventies that the antiseptic doctrine which Lister had enunciated in 1867 was recognized and, even then, quite indifferently and skeptically applied in current practice. Consequently, the practice of surgery was largely circumscribed to the extremities in which fractures, dislocations, amputations, the ligation of arteries and the treatment of ulcers and wounds constituted the chief work of the surgeon. The limitations of surgery were indeed then very great and, as late as 1881, did not constitute more than 3.2 per cent of the total medical treatment given by the hospital. There was then no abdominal or gynecologic surgery, no thoracic surgery, no orthopedic or neurologic surgery; nothing except fractures, dislocations and amputations; lithotomy or cutting for stone; an occasional trephining; rarely a celiotomy for ovarian tumor, strangulated hernia, or an operation for cataract, to divert the attention of the surgeon away from the extremities or to extend the excursions of surgery beyond them. And this was the status of surgery throughout the world until the last quarter of the nineteenth century, when the genius of Pasteur and Lister emancipated surgery and suddenly expanded its dominion and its therapeutic resources to its present limitless horizon. It is not surprising that the surgeons of the later eighteenth and of the nineteenth centuries should have cultivated a special interest in the surgery of the blood vessels which provided the most inviting field for the display of anatomic knowledge and for the accomplishment of daring feats in surgery which, at that time, were justly regarded as the highest attainments of the art. The surgeons who attained the greatest fame throughout the world had won their reputations largely through their achievement in the surgery of the blood vessels. The great names of Desault, Dupuytren, Anel, Deschamps, Brasdor, Lisfranc, Velpeau and Broca, in France; Valsalva, Lancisi, Scarpa and Porta, in Italy; John and William Hunter, Charles and John Bell, Abernethy, Guthrie, Sir Astley Cooper, Liston, Hodgson, Syme, Wardrop, Billingham and Compton, in the British Isles; Graeffe, Purmann, Dieffenbach, Stromeyer, Langenbeck and Hütter, in Germany; Pirogoff in Russia. In America, the

daring and success of the early New England and Eastern surgeons in the ligation of the carotids, subclavians, iliacs, femorals, etc., were far in advance of the European surgeons; conspicuous among these were Wright Post, W. F. Cogswell, Amos Twitchell, J. S. Dorsey, James McGill, W. Gibson, Willard Parker, and above all, Valentine Mott*—*facile princeps* of the vascular surgeons of his time—are inseparably bound with the most notable achievements of the preanesthetic and prelisterian period. It is natural, therefore, that the surgeons and teachers at the Charity Hospital living in the same period should have yielded to identical aspirations and tendencies that dominated the surgical world and that incited them to devise and perform operations for the cure of aneurysms which were new and daring for that age. Thus, we see the first successful ligation of the common iliac artery for aneurysm performed at the Charity Hospital, in the early thirties, by Charles Luzenberg (1808–1848), the first professor of surgery in Louisiana; and again by his successor, Warren Stone (1808–1872), who was the first to use a metallic ligature (silver wire) to occlude the same vessel, in order to avoid the dangers of the classic, but septic, silk ligatures. Later, Wederstrandt repeated the same feat, and Wedderburn ligated the third subclavian, while Stone successfully attacked the aneurysms of the extremities, including traumatic aneurysms of the gluteal and vertebral arteries, in the forties and fifties. Other ligations were performed in the same period by Picton, Armand Mercier, Daret, Compton, Choppin, Schuppert, and still others, too numerous to mention. But the crowning event in the prelisterian period of the hospital's history was the first successful ligation of the innominate for a subclavian aneurysm by Andrew Smyth in 1864—which gave this illustrious surgeon, and the hospital, an enduring and widespread international fame.

The descriptions of these operations, as they were performed by these enterprising pioneer surgeons, in the difficult and unprepared conditions of surgical technic which prevailed in the preantiseptic period, read to the surgeon of to-day as a sort of adventurous epic well tinctured with heroic flavor.

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* In his memoir of Mott (1785-1865), published in 1868, Professor S. D. Gross writes: "No Surgeon, living or dead, ever tied so many vessels or so successfully, for the cure of aneurysm, the relief of injury or the arrest of morbid growths. The catalogue, inclusive of the celebrated first ligation of the innominate artery (1818) (succumbed to secondary hemorrhage on the 28th. day), comprises eight examples of the subclavian artery, 51 of the primitive carotid, two of the external carotid, one of the common iliac (first successful) 1827, six of the external iliac, two of the internal iliac, 57 of the femoral and ten of the popliteal; in all, 138." Besides the ligations, he had 165 lithotomies and over 1,000 amputations to his credit.

Sir Astley Cooper, one of his early teachers, spoke of him as "the surgeon who had performed more of the great operations of surgery than any man living, or that ever did live." (Gross' Memoirs, l. c., and in Century of Am. Surgery, Lea, Phila., 1876; F. Garrison, Bulletin N. Y. Academy of Med., August, 1925, 2nd. series 1.)

It was my good fortune to begin my internship in the Charity, at the very dawn of the listerian era (1877), but I can well remember a number of amputation stumps with long, dangling ligatures attached like suppurating setons to the main arteries, as a sign that the short, buried, antiseptic ligature had not yet won the confidence of all surgeons.

Even in the seventies and early eighties, the fear of infection, suppuration and secondary hemorrhage which was an almost invariable sequela of the ligature for the cure of aneurysm, awed the surgeons and restrained them from any open attack on the aneurysms of the great vessels and led them to resort, when at all possible, to mechanical and bloodless methods such as indirect digital compression (Jonathan Knight, in the United States, and Belmas, in France, 1844); instrumental compression by various devices (W. Reid, 1875); flexion (Hart, 1857); needling, malaxation and wiring for the inoperable central aneurysms.

I had assisted as an undergraduate intern in the application of all these methods by my distinguished chiefs—T. G. Richardson, Samuel Logan, A. B. Miles, and particularly Dr. Edmond Souchon, whose valuable contributions to the surgery of aneurysms have left an enduring mark in the literature. It was only when the bloodless methods failed that the hunterian ligature was adopted, usually, as a last resort. Incision of the sac after bipolar ligation and packing (Antyllus), or excision of the sac were not thought of except when rupture was threatened by infection and suppuration or sloughing, and even then an amputation was deemed safer by most of the staff. In the aortic and other central aneurysms, absolute rest in bed with a restricted diet and the administration of mercury, potassium iodide, opium or morphia for pain, and such circulatory depressants as veratrum viride, or aconite, following the ancient teachings of Valsalva, Jeliffe Tuffnel and Billingham of the Irish school, was the only treatment—a mode of treatment which has not materially improved up to the present except for the benefits of the arsenical preparations, salvarsan, arsphenamine and bismuth, which undoubtedly exercise a prophylactic beneficial influence in arresting the aortitis or arteritis of syphilitic subjects when timely administered before the actual aneurysmal stage is reached.

In this connection, the rise and spread of the *pathogenic aneurysms* as a manifestation of arterial disease, particularly involving the aorta and its large central branches—when coupled with their greatly increased prevalence in the Negro race since emancipation—is one of the most striking features of the Charity Hospital experience.

It is a well-established fact that the aneurysms of the aorta were a relatively negligible factor in the mortality of the Southern states before the Civil War, with even less incidence in the Negro slave population.*

* For a full description and appreciation of the great value and importance of the Charity Hospital as a center for the study of the comparative pathology of the white and Negro races, see the author's monograph on "The Surgical Peculiarities of the American Negro. A statistical inquiry based upon the records of the Charity Hospital." Trans. Am. Surg. Assn., **14**, 130, 1896.

At the Charity Hospital, during the period 1825-1831, only two aneurysms were recorded in 15,707 total admissions, or one aneurysm to 7,853 admissions. In the decade 1865-1875, 65,935 total admissions yielded 63 aneurysms, or 1:1,046 admissions. In the decade 1884-1893, a total of 199 aneurysms were admitted; whites 143 (71.86 per cent), or 1:312.5, and colored 56 (28.14 per cent), or 1:344.8. In 1904-1931 (27 years) a total of 1027 aneurysms were admitted, or 1:335 surgical admissions; 1:1,166 total white admissions and 1:329.9 total colored admissions.

In the five years, 1935-1939, a total of 412 aneurysms were admitted, or 1:742 total admissions and 1:296 surgical admissions. In this group the ratio was one white to 3.5 Negroes. Of these 412 aneurysms, 90 per cent were pathogenic, chiefly aortic, and of these, 73.3 per cent were Wassermann positive. Of the total 412, only 45, or 10.9 per cent, were regarded as surgical and were operated upon.

It is worthy of note that, while the surgical or operative cases have not kept pace with the enormous increase in the "medical aneurysms" (the pathogenic, inoperable, chiefly aortic, aneurysms), the statistics of the operated cases show the extraordinary improvement that has been wrought in the results of the operations by modern methods. During the 52 years, 1826-1877, (including the preanesthetic and preantiseptic periods), 175 aneurysms were admitted for treatment at the Charity Hospital. During this period the methods of ligation and bloodless methods of indirect compression were in vogue. There were in all 68 deaths, or a gross mortality of 45.3 per cent. Collective staff statistics of Charity Hospital from the files in the record library show that, from August, 1905 to July, 1934 (29 years), 234 patients were operated upon for aneurysms by 37 surgeons (including 101 by Dr. Matas). Different methods were employed, including ligation, extirpation and endo-aneurysmorraphy. There were 38 deaths, or a gross mortality of 16.2 per cent.

The result of this inquiry shows: (1) That aneurysm as a disease and not trauma, has increased 13 times over its incidence 100 years ago; (2) that this increased prevalence is particularly apparent in the colored population (3½ times more prevalent in the Negro); (3) that this increased prevalence is due to the vast preponderance of the pathogenic aneurysms of arterial disease (90 per cent), while the traumatic aneurysms have gained very little in prevalence in the last 40 years (ten pathogenic to one traumatic); (4) that fully 75 per cent of the pathogenic aneurysms were aortic or central; (5) that 73.3 of these occur in Wassermann positive subjects; and (6) apart from the increased incidence of aortic aneurysms due to roentgenographic studies and greater facilities for diagnosis, there has been an actual increase in the prevalence of the disease, confirmed by postmortem evidence.* That, while the incidence of aneurysms has vastly increased in the last 60 years, it has been chiefly in the aortic, internal or medical aneurysms, with very little increase in the operable or surgical peripheral aneurysms, since we have had no condition of warfare which would increase traumatic aneurysms.

As far as the arteriovenous aneurysms are concerned, the old teaching regarded them as relatively benign and not liable to rupture—a tradition which lingered from the days of William Hunter (1774); they were allowed to remain undisturbed except by purely mechanical methods of compression. When complicated by large varicose sacs, by infection or by other disabling varicosities they were attacked by the open method, only as a last resort. When surgery was required, the proximal artery was ligated, but, as experience too frequently proved, with no cure and often gangrene of the limb. It

* Ref. I. I. Lemman. Am. Jour. Med. Sc., vol. 105, No. 5, 1916.

was only after the great treatises of Paul Broca, in the fifties, and Pierre Delbet and von Bramman, in the eighties, that the principles underlying the cure of arteriovenous aneurysms were clearly understood and that the extirpation of the fistulous segments or the quadruple ligature were more frequently resorted to.

None the less, the complete ignorance of the profession regarding the secondary and evil effects of arteriovenous fistula upon the heart and circulation which came as a revelation with the vast experience of the World War, continued until comparatively recent times, to protect these aneurysms from active and aggressive surgical action. We now know that every arteriovenous fistula of large size and which involves the greater blood vessels is, apart from its disabling local and regional effects, a perpetual menace to the integrity of the circulation, and that sooner or later, according to the size of the fistula and other correlated factors, the heart will suffer serious organic changes—dilatation, myocardial degeneration with a terminal fatal decompensation.

* * * * *

Going back to my personal experience, I would state that my internship in a hospital where the surgery of the blood vessels had become a proud historic tradition, my association with the great surgeons and teachers just mentioned, who were especially concerned with the cure of aneurysm, and the anatomic experience that I had acquired early in my career as demonstrator of anatomy for over ten years in the dissecting rooms of the medical school, all combined to give me a special interest in vascular pathology and thereby to utilize the unusual opportunity given me to study, clinically and surgically, the ever fascinating problems that for practically 60 years have presented themselves to me as a visiting surgeon of the Charity Hospital and of the other local institutions with which I have been associated.

In the course of these years the opportunities for the treatment of aneurysm and other vascular lesions and diseases rapidly increased and, as my special interest and experience in the treatment of these cases became known to my associates, pupils and friends, I was favored by their kindness in referring many in and out of town patients, some from distant states, who increased my statistics out of proportion to the number that would have been furnished to any single operator by the local surgical population. In earlier years I operated upon a number of patients in private houses, but, as our hospital facilities increased I operated almost exclusively in the various hospitals with which I was connected. (In addition to the Charity Hospital, my list includes operations performed at the Hotel Dieu; New Orleans Sanitarium, which became the Presbyterian Hospital; the Eye, Ear, Nose and Throat Hospital; and Touro Infirmary.) After 1904 my practice was confined to the Charity Hospital and the Touro Infirmary.

THE FIRST ENDO-ANEURYSMORRAPHY*

Prior to 1888, I treated the aneurysms and other allied vascular lesions that came under my care by the classical methods in vogue at the time; and in these the ligation on the hunterian or Anel principles predominated. But, on April 6, 1888, I operated upon a young male Negro for a very large traumatic (multiple gunshot) aneurysm of the brachial artery, extending from the armpit to the elbow, which opened my eyes to the possibilities of an entirely new method of conservative treatment which was to revolutionize my previous notions of aneurysmal surgery. In this case, the successive ligation of the main artery on the proximal and distal poles of the aneurysm had been followed by relapse, and it seemed to me, then, that I had no other alternative but to extirpate the sac. When I exposed the sac and emptied its contents, the failure of the ligations to control the circulation was easily explained by the appearance at the bottom of the sac of three large orifices corresponding to the collateral branches which opened into the sac in the segment of the artery included between the ligatures. It was evident that it was these collateral orifices that fed the sac despite the ligatures that had been placed at each one of its poles. I, at first, intended to secure these collaterals by excising the sac, but the branches of the brachial plexus of nerves were so densely incorporated in its walls that I could not have dissected them out and detached them, without serious damage, thereby paralyzing the arm. It occurred to me then that the easiest way out of this awkward dilemma was *to seal the orifices of all the bleeding collaterals by suturing them as we would an intestinal wound*, leaving the sac attached and undisturbed in the wound. This procedure was at once put into effect and the hemostasis was so perfect and satisfactory that it seemed to me strange that no one should have thought of so simple an expedient before.

This, then, was my first case of aneurysm treated by the method of intrasaccular suture which, with further experience and thought, I developed and systematized into the method of endo-aneurysmorraphy or intrasaccular suture, and which, in its three phases—the obliterative, the restorative and the reconstructive—is now associated with my name. My initial case was reported in the Philadelphia Medical News, October 27, 1888—now more than 51 years ago. But the first systematic description of the method was not reported until 14 years after this publication, 1902, at the meeting of the

* The spelling of the word "endo-aneurysmorrhaphy" is in accordance with the classical orthography of the dictionaries. This neologism was originally coined out of the Greek by me, in 1902, as descriptive of the eponymic designation "Matas operation" by which it was usually referred to in the literature. Many have adopted "endo-aneurysmorraphy" and others simply "aneurysmorraphy." Personally, I have abandoned the original form for the contracted "endo-aneurysmoraphy" or "endo-aneurismoraphy" which is equally descriptive and faithful to its origin, but much simplified by the elimination of unnecessary consonants, following the suggestion of the Carnegie Spelling Board. When the term is frequently repeated, the word is still further abbreviated by writing "endo-a," as in endo-a, *obliterative;* endo-a, *restorative;* endo-a, *reconstructive.*

American Surgical Association held in Albany that year, and published in the ANNALS OF SURGERY for February, 1903—now 37 years ago.* The appearance of this paper was followed by many published reports and personal communications from other surgeons at home and abroad who confirmed the practical simplicity and conservative value of the operation and gave it their commendation and support. Since the original report in 1902, I have published a number of statistical summaries which included my personal operations and the cases reported by other surgeons as these steadily increased and accumulated in the literature. Of these statistical papers, the most complete are: The report of 225 operations presented by invitation at the International Medical Congress held in London in 1913; the second is my Mutter lecture in Philadelphia, 1915 (289 cases); the third is my report to the French Surgical Congress held in Paris in 1922, when I reported 350 aneurysmorraphies, including my own personal cases. Again, in October, 1925, at the Clinical Congress of the American College of Surgeons, in Philadelphia, I was able to increase the collective experience in aneurysmorraphy to 478 recorded operations contributed by the surgeons all over the world. Of these, 65 had been performed in my own practice, including the arterial and arteriovenous aneurysms. Altogether, the surgeons of Louisiana, including my own cases, had recorded over 110 aneurysmorraphies in all three of its modes, and of which approximately 80 per cent are of the obliterative type.

The collective results of these operations as they appear in the detailed summaries of my London (1913), Mütter lecture, Philadelphia (1915), Paris (1922), and Philadelphia (1925) statistics are, on the whole, very gratifying, and statistically superior to those of other methods resorted to for the same class of cases.

Since 1927, when the list of these operations, collected from the literature, had increased to 505, I have made no systematic search or compilation of cases reported in the literature. In fact, the operation has entered so largely into the current surgery of most countries that it has ceased to be a novelty and is reported only in exceptional cases.

Taken altogether, in all its modalities or phases, the restorative, the reconstructive, and the obliterative—the mortality (exclusive of the aortic aneurysms) did not exceed 4.5 per cent; the gangrene following operations for aneurysms of the extremities, 3.5 per cent; secondary hemorrhage, 1.6 per cent; and the relapses, chiefly in reconstructive cases, 1 per cent of the 350 operations reported to the French Congress in 1922. A far better showing, as a whole, than the statistics of the ligature or of extirpation which were so exhaustively compiled by Delbet and Mocquot, and by Monod and Van-

* In the interval between these years, further clinical and anatomic opportunities presented themselves for the study of aneurysmal sac-interiors, which led to an entirely new study of the morphology and variations in the anatomy of aneurysmal sacs, as fully described under "Morphology of the Sac" in my treatise on aneurysms, in Keen's Surgery. This study led to the subdivision of endo-aneurysmorraphy into its three varieties, as previously stated.

verts in France in 1911, and later by the German and Austrian surgeons during the World War as shown in my chapter on the "Military Surgery of the Vascular System," in Keen's Supplemental War-Volume VI, 1921. (See ref. 44 this Bibliography)

In my statistics of 505 operations, which I had compiled from all sources up to 1927, I did not include the operations performed by the German and Austrian surgeons during the World War. Nearly 50 per cent of the cases of traumatic aneurysms reported by these surgeons as treated by the methods of intrasaccular suture, were *lateral* arteriorraphies performed in aneurysmal sacs or mature hematomas and in this way were virtually, in principle and technic, my restorative endo-aneurysmorraphies,* which I had described long before the war, but never credited, in Germany, until quite recently.

In the above statistics I have only referred to the operations performed by the method of aneurysmorraphy or intrasaccular suture, but I would like to make it clear that I do not treat all aneurysms by this or any one method. I do not apply endo-aneurysmorraphy to all aneurysms; on the contrary, I am decidedly eclectic, selective and discriminative in my practice, and this attitude is well shown by the following summary of my personal experience up to the present time.

CLASSIFIED SUMMARY OF 620 OPERATIONS UPON THE BLOOD VESSELS, PERFORMED FOR ALL CAUSES, BY RUDOLPH MATAS, M.D., NEW ORLEANS, LA., BETWEEN THE YEARS 1888 AND 1940 †

PART I

ANATOMIC DISTRIBUTION

Operations upon the Common Carotid for Aneurysms or Aneurysmal Conditions: (35 proximal, 18 distal); (proximal—24 arterial, 11 arteriovenous)	53
Operations upon the Common Carotid for Conditions other than Aneurysms	25
Operations upon the Internal Carotid (including the middle cerebral): (4 arterial, 3 arteriovenous)	7
Operations upon the External Carotid and its Branches for Aneurysms or Aneurysmal Conditions: (5 arterial, 6 arteriovenous)	11

* Bier, A.: On War Aneurysms. Handbuch Arztliche Erfarungen in Welt Kriege. Bd. 1. Chirurg. 2nd. Part, 448–491, 1922; and C. Franz. Lehrbuch d. Kriegschirurgie. 2nd. Edit., 1939.

† This statistical summary is based on the author's classified tables of individual cases in which the essential facts are given in tabloid form for each patient, numbered in the order of his admission to the hospital or in the author's private records.

It is planned by the author to publish these tables conjointly with the descriptive text, in a series of papers devoted to the discussion of the material, and the conclusions drawn from his experience, in each of the regional or anatomic groups, as outlined in this summary.

Operations upon the External Carotid and its Branches for Conditions other than Aneurysms 321

Total Carotid Operations 417

Innominate:
6 provisional ligations (2 arterial, 4 arteriovenous)
4 permanent ligations (all arterial)
3 wiring with Colt's apparatus (all arterial) 13

Subclavian Aneurysms or Wounds: (29 arterial, 7 arteriovenous) 36

Axillary: (1 arterial, 3 arteriovenous, 4 injuries in the course of radical extirpations of malignant growths) 8

Brachial: (3 arterial, 2 arteriovenous) 5

Radial: (3 arterial) 3

Hand: (1 arterial, 1 arteriovenous) 2

Abdominal Aorta: (1 ligation, 3 wired, 3 explorations with nothing done for aneurysm) 7

External Iliac and Iliofemoral: (Iliofemoral—14 arterial, 4 arteriovenous; external and common iliac—3 arterial, 1 arteriovenous; 1 for injury in radical extirpation of femoral malignancy) 23

Internal Iliac: (4 bilateral ligations as first stage of Wertheim operation) 8

Femoral: (14 arterial, 18 arteriovenous) 32

Femoropopliteal: (9 arterial, 3 arteriovenous) 12

Popliteal: (42 arterial, including arterial accessory sac in an arteriovenous aneurysm; 4 arteriovenous; 1 circular angiorraphy for myxofibroma; 2 amputations for popliteal injury, gangrenous on admission) 49

Tibial and Pedal: (4 arterial, 1 arteriovenous, peroneal) 5

Total 620

OPERATION FOR ANEURYSMS, ANEURYSMAL LESIONS (ANGIOMATA, CIRSOIDS ETC.), AND WOUNDS

Common Carotid:
35 proximal occlusions (24 arterial, 11 arteriovenous)
18 distal occlusions (all arterial) 53

Internal Carotid (including middle cerebral):
(4 arterial, 3 arteriovenous) 7

External Carotid and Branches: (5 arterial, 6 arteriovenous) 11

Innominate:
6 provisional ligations (2 arterial, 4 arteriovenous)
4 permanent ligations (all arterial)
3 wiring (all arterial) 13

Subclavian: (29 arterial, 7 arteriovenous) 36

Axillary: (1 arterial, 3 arteriovenous) 4

Brachial: (3 arterial, 2 arteriovenous) 5

Radial: (all arterial) 3

Hand: (1 arterial, 1 arteriovenous) 2

Abdominal Aorta: (all arterial) 7

External, Common Iliac: (3 arterial, 1 arteriovenous) 4

Iliofemoral: (14 arterial, 4 arteriovenous) 18

Femoral: (14 arterial, 18 arteriovenous) 32

Femoropopliteal: (9 arterial, 3 arteriovenous) 12

Popliteal: (44 arterial, 4 arteriovenous) 48

Tibial and Pedal: (4 arterial, 1 arteriovenous, peroneal) 5

Total 260

OPERATIONS FOR CONDITIONS OTHER THAN ANEURYSMS

Common Carotid	25
External Carotid and Branches	321
Axillary	4
External Iliac	1
Internal Iliac (4 bilateral)	8
Popliteal	1
Total	360

RECAPITULATION

Operations for aneurysms, aneurysmal lesions and wounds	260
Operations for conditions other than aneurysms	360
Total	620*

PART II

REGIONAL CLASSIFICATION IN DETAIL; WITH RESULTS

SURGERY OF THE CAROTID VESSELS

Operations upon Common Carotid (occlusions with aluminum bands, ligatures, sutures) for Aneurysms or Aneurysmal Conditions:	
42 arterial aneurysms: 24 proximal occlusions (cardiac side) 18 distal occlusions†	
11 arteriovenous aneurysms: including 6 intracranial, carotid cavernous aneurysms: 2 without exophthalmos, and 4 with exophthalmos	53
Operations upon Internal Carotid (band occlusions or ligatures):	
5 arterial aneurysms, including wound of middle cerebral	
2 arteriovenous intracranial aneurysms, without exophthalmos	7
Operations upon External Carotid and its Branches for Aneurysms or Aneurysmal Conditions (ligatures):	
4 arterial aneurysms	
7 arteriovenous aneurysms, including 2 intracranial aneurysms without exophthalmos	11
Total operations for carotid aneurysms	71
Operations upon the Common Carotid (ligatures or band occlusions) for Non-aneurysmal Lesions	25
Operations upon the External Carotid and its Branches for Conditions other than Aneurysms: (ligatures)	
90 ligations of external carotid trunk (including 8 bilateral ligations)	

* Since this list was completed (May 1, 1940), two more aneurysms have been added. Both popliteals. One a traumatic, arteriovenous; treated by excision and quadruple ligature, with complete recovery and perfect functional result, and one arterial, atheromatous. Obliterative endo-aneurysmorraphy complicated by popliteal thrombosis. Recovery; with amputation of foot.

† Distal occlusions chiefly on the Brasdor-Guinard principle for innominate and ascending aortic aneurysms involving origin of innominate. In these cases, the common carotid and right subclavian were ligated at the same, or more often at separate sittings, always beginning with the common carotid first, and applying a *removable* aluminum band (Matas-Allen type), to test the efficiency of the collateral circulation in the brain. For history and technic of these bands, ref. bibliography Nos. 23, 25, 28, 34, 44, 83 and 97.

34 ligations of individual branches of the external carotid in 34 of the above 90 ligations

197 thyroid ligations preliminary to thyroidectomies 321

Total carotid operations for nonaneurysmal conditions 346

RECAPITULATION

Total carotid operations for aneurysms 71
Total carotid operations for conditions other than aneurysms 346

Total carotid operations for all causes 417*

REMARKS.—As shown in the preceding tables, the occlusions of the common carotid (by bands or ligatures) for aneurysms amount to 53 cases. In addition to this, there are 25 common carotid occlusions for nonaneurysmal conditions, making a total of 78 common carotid ligations for all causes. If to these 78 common carotid ligations we add seven ligations of the internal carotid for aneurysm, the combined ligations of the common (78) and the internal carotid (seven) will amount to a total of 85 band occlusions or ligations of the two main carotid trunks for all causes, exclusive of 101 ligations of the external carotid for all causes, and 231 ligations of the external carotid branches.

SURGERY OF THE COMMON CAROTID

Proximal Occlusions:

For arterial aneurysms:

20 bands, with one death from cerebral complications in four days†
4 ligations, no deaths.

For arteriovenous aneurysms:

8 bands, with one death in nine days of heart complications (angina).
(One patient with cerebral symptoms was rescued by timely removal of band and later cured by band after long periods of compression.)
2 transvenous sutures, with one death in six days, of angina.[a]
1 intrasaccular suture, cured.

35 Total proximal occlusions, with two deaths. Mortality 2:35, or 5.7 per cent.

[a] Same patient.

Distal Occlusions (on the Brasdor-Guinard procedure for innominate and aortic aneurysms) all arterial:

14 bands, no operative deaths.‡
4 ligations, no operative deaths.¶

18 Total distal occlusions, no operative deaths.

* Ref. bibliography Nos. 19 (carotids), 23, 28, 41 and 44.

† One patient died suddenly at his home, two months after band occlusion of common carotid, of hemorrhage from rupture of retropharyngeal abscess causing erosion and infection of old aneurysmal sac.

‡ Two patients died of progressive tracheal compression from aneurysm, each about three weeks after distal occlusion of common carotid.

¶ While attempting to perform a sternal resection to expose aneurysm, 19 days after distal ligation of common carotid, one patient died of laryngeal spasm, under ether.

Operations upon the Common Carotid or Conditions other than Aneurysm:

12 bands, all with operative recovery. (Two patients with cerebral symptoms were saved by timely removal of band.)

8 ligations, with three deaths from cerebral complications.

5 excisions of artery with tumor, with one death from cerebral complications.

25 Total, with four deaths. Mortality, 4:25, or 16 per cent

TYPES OF OPERATION IN SURGERY OF THE COMMON CAROTID

Bands:

40 proximal occlusions with bands, (three deaths)
- 20 for arterial aneurysms.
- 8 for arteriovenous aneurysms.
- 12 for conditions other than aneurysms.

14 distal occlusions with bands, no operative deaths 54

Ligatures:

4 proximal ligations for aneurysms, no deaths.

8 proximal ligations for conditions other than aneurysm, three deaths.

4 distal ligations, no operative deaths 16

Suture Methods: No operative deaths.

2 transvenous sutures for arteriovenous aneurysms.

1 intrasaccular suture for arteriovenous aneurysm 3

Excisions:

5 for conditions other than aneurysms, one death 5

Total 78

SURGERY OF THE INTERNAL CAROTID*

3 bands to internal carotid for arterial aneurysms:
- 1 for aneurysm of internal carotid.
- 1 for subclavian aneurysm (test bands), as precaution before ligating the innominate.
- 1 for aneurysm of left common carotid at bifurcation. Death two days later, in spite of removal of band on appearance of cerebral signs, 18 hours after occlusion.

3 ligations, one for arterial aneurysm and two for arteriovenous aneurysms, of internal carotid, in all of which bands to common carotid had failed to cure the aneurysm. No deaths.

1 emergency hemostatic intracranial control (middle cerebral) by forceps. Death of preoperative hemorrhage and shock.

7 Total occlusions of internal carotid, with two deaths. Mortality, 2:7, or 28.5 per cent

REMARKS.—In addition to the above, two arterial extracranial aneurysms of the internal carotid were cured by bands placed upon the common carotid; making in all a total of seven aneurysms of the internal carotid, of which four followed tonsillectomies or tonsillar abscesses.

A summary of intracranial aneurysms follows, the operated cases also appearing previously in their respective tables.

* Ref. bibliography Nos. 88, 91 and 97.

INTRACRANIAL ARTERIOVENOUS FISTULAE OF THE CAROTID TRACT*

I. Extracranial bands on the common carotid for intracranial arteriovenous fistulae without exophthalmos 2
II. Ligations of the internal carotid for intracranial arteriovenous fistulae without exophthalmos after failure of common carotid ligations 2
III. Extracranial bands on the common carotid for carotid-cavernous aneurysms (pulsating exophthalmos) 4
IV. Previous ligations of the common carotid for pulsating exophthalmos followed by relapse and ultimate cure by spontaneous thrombosis 2
V. Case of spontaneous carotid-cavernous arteriovenous aneurysm (pulsating exophthalmos) cured by spontaneous thrombosis without operation 1
VI. Cases of intracranial arteriovenous communication (2 with exophthalmos, 2 without exophthalmos), in which for various reasons no operation was performed, and treatment limited to systematic carotid compression 4

TOTAL (not including cases observed and treated with colleagues) 15

SURGERY OF THE EXTERNAL CAROTID AND ITS BRANCHES†

Operations upon the External Carotid and Its Branches for Aneurysms or Aneurysmal Lesions:

Arterial: (no deaths)

3 ligations, 1 of the external carotid and 2 of the meningea media.
1 excision of arterial varix (scalp).

Arteriovenous: (no deaths)

5 ligations, 3 for cavernous angiomata of the scalp and ear.
1 obliterative endo-aneurysmorraphy.
1 quadruple ligation.

11 Total, all cured

Operations upon the External Carotid and its Branches for Conditions other than Aneurysms (mainly malignant grows of the jaws, mouth, neck, *etc.*):

90 ligations with seven deaths. Mortality, 7:90, or 7.7 per cent.
34 of the above 90 cases in which the branches of the external carotid were separately ligated.
197 thyroid ligations.

321 Total

REMARKS.—From the above table it will be seen that I have recorded 101 ligations of the external carotid, of which 11 were ligations for aneurysms and aneurysmal conditions in the area of the external carotid distribution, all followed by recovery without cerebral complications.

In addition to these 11 ligations, there are 90 ligations of the external carotid for surgical conditions other than aneurysms; included in this were eight bilateral ligations of the external carotid preliminary to operation for very extensive malignant disease. In the majority of cases the external carotid and its main branches were ligated together with the main trunk, or this, in a few instances, was injected with paraffin following the Dawborn pro-

* Ref. bibliography: Nos. 83, 88, and 97.

† Ref. bibliography, No. 19, aneurysms of special arteries (carotids).

cedure, to obtain as complete an occlusion or obliteration of the external carotid tract as possible.

These 90 ligations of the external carotid were followed in nine cases by postoperative complications which proved fatal in seven patients. The postoperative complications are classified as follows: Cerebral, five; pulmonary, three; cardiopulmonary, with a question mark (supposed to have been cerebral, but autopsy proved negative for cerebral lesions), one.

Of the five cases in which the cerebral complications (stupor, contralateral hemiplegia, aphasia, coma) followed the ligation of the external carotid, three proved fatal and two recovered. But of these five cases of cerebral complications, only three can be clearly and positively attributed to the ligation of the external carotid. In the other two, one of the patients (age 55; resection of right upper jaw for sarcoma) had undergone the ligation of the external carotid without cerebral disturbances until the ninth day, when secondary hemorrhage compelled the ligation of the common carotid by the resident house surgeon. This was followed promptly by stupor, hemiplegia and aphasia from which the patient finally recovered, with seeming cure also of the disease. The other patient (age 44; bilateral ligation of the external carotid preliminary to excision of the tongue for extensive carcinoma with multiple, diffuse, secondary lymph node metastases in the submaxillary regions and neck) was operated upon in two stages. The last operation on the nodes was followed by aphasia and other signs of a right-sided block of the internal carotid, two weeks after the bilateral ligation of the external. The cerebral signs coincided with a purulent infection in the submaxillary wound. Despite this, the patient gradually recovered from the operation, the brain symptoms clearing completely after a few months. The patient died nevertheless, three years later, from generalized cervical and mediastinal metastases.

It would seem from this, that our experience in the ligation of the external carotid for nonaneurysmal causes were followed by cerebral complications in three cases, two of which proved fatal, thus establishing an incidence of cerebral complications in the nonaneurysmal group of 3:90, or 3.3 per cent, and a fatality of 2:90 or 2.2 per cent; or a death rate of 2:3, or 66.6 per cent of the brain complications.

If the 11 ligations for aneurysmal conditions are added to the 90 ligations for nonaneurysmal causes, equaling in all a total of 101 ligations, the incidence of the cerebral complications after this special ligation would be reduced to 2.97 per cent, and the mortality from cerebral complications would be reduced to 1.99 per cent.

From this we gather that the ligation of the external carotid must continue to be regarded as a relatively benign ligation, especially when compared with the dangers of the internal and common carotid ligations. None the less, an incidence of 3 per cent cerebral complications makes us regard the ligation of the external carotid as an added risk to whatever other dangers may be inherent in the operation for which the ligation is performed; and

this risk, minimal as it is, must weigh against the benefits that are expected to accrue from the ligation. I would say that, personally, I would not allow this possible risk to outweigh the positive and, undoubtedly, great hemostatic advantages that the preliminary ligation of the external carotid affords in controlling the vascular areas involved in the field supplied by these arteries.

Danger of Thrombus at the Bifurcation.—When the first death from a preliminary ligation of the external carotid (ligation of the external carotid preliminary to excision of the second and third divisions of the trigeminus at the basal foramina, employing Mixter's modification of Salzer's method) occurred in my practice in 1893, I was very much impressed with the danger of a ligation close to the bifurcation, and believed that a thrombus, starting at the ligation and extending to the bifurcation, would in all probability break off at the tip to be swept away to the brain by the force of the carotid stream. I still believe this is possible, and perhaps probable, as long as the internal carotid is pervious. Once the internal carotid is firmly plugged by the clot, the possibility of embolism ceases, as the arterial pressure will drop beyond the obstruction, and the carotid stream will be reversed from the brain to the seat of the obstruction, making it impossible for any emboli to float to the circle of Willis; though it is possible, as has been demonstrated by postmortem evidence, that a thrombus or clot may extend all the way from the bifurcation (after internal carotid ligations) to the level of the cavernous sinus in the internal carotid.

After my first experience I always ligated the external carotid as far as possible from the bifurcation, consistently with the hemostatic or denutrient purpose of the ligation. When the superior thyroid originated close to the bifurcation, I ligated the thyroid at its origin and the external carotid beyond this point. The very frequent anomalies in the origin of the anterior and ascending branches of the external carotid compelled variations in the ligation of the parent trunk and its branches in my efforts to keep at a respectable distance from the bifurcation. My experience shows that, despite these anatomic precautions, cerebral ischemia may occur with or without a detached embolus. None the less, it is rational to ligate as far as possible beyond the bifurcation. In any case, it would seem that the thrombus must precede the embolus, if this is ever detached, before the internal carotid has been completely plugged.

SURGERY OF THE INNOMINATE*

6 provisional ligations for hemostatic control during operations for subclavian aneurysms: (no deaths)
2 arterial.
4 arteriovenous.
4 permanent occlusions of the innominate with bands or ligatures: (3 bands, 1 ligation, all arterial)
There were two deaths, one from pulmonary complications and hemorrhage on

* Ref. bibliography Nos. 19 (section on aneurysms of special arteries, innominate), 71 and 83.

the sixth day after band occlusion; another (ligation) from consecutive secondary hemorrhage and cerebral complications from infected gunshot wound of the carotid.

3 innominate aneurysms wired with Colt's apparatus, with notable effect on the aneurysms and marked symptomatic relief. In these cases wiring followed distal ligation of the common carotid and third right subclavian as a precaution against cerebral embolism.

13 Total, with two deaths. Mortality, 2:13, or 15.3 per cent

SURGERY OF THE SUBCLAVIAN*

Operations upon the Subclavians for Arterial Aneurysms: (no deaths)

12 proximal bands or ligations for subclavian aneurysms.
Right subclavian, 3 bands, 2 ligations.
Left subclavian, 3 bands, 4 ligations.

2 ligations of third subclavian for axillary traumatic (pulsating) hematomata.
Right subclavian, cured.
Left subclavian, died of preoperative shock and hemorrhage.

11 distal occlusions of the third subclavian for innominate and aortic aneurysms, 8 bands, 3 ligations (no deaths).

Operations upon the Subclavians for Wounds:

2 for subclavian wounds:

1 clamping for wound of inferior thyroid in extirpating metastatic carcinoma of the larynx and thyroid. (Died third day from shock, surgical anemia and hypostatic pneumonia.)

1 excision of cervical rib and segment of blocked artery for thrombotic occlusion of left subclavian artery.

2 for wounds of subclavian branches:

1 traumatic (bullet wound) aneurysm of vertebral artery; direct intervention by aneurysmotomy and packing vertebral canal, cured.

1 emergency hemostasis of internal mammary (with clamp) for stab wound of internal mammary causing fatal hemothorax, died.

29 Total operations on subclavian artery and branches, three deaths. Mortality, 3:29, or 6.9 per cent

Operations upon the Subclavians for Arteriovenous Aneurysms:

6 direct operations:
Right subclavian, 4 ligations, no deaths. In one of these, the dilated subclavian artery was ligated to control an arteriovenous angioma of the arm.
Left subclavian, 1 band and 1 transvenous suture, no deaths.

1 ligation of thyroid axis and branches for arteriovenous aneurysm after provisional ligation of innominate, cured.

7 Total operations upon the subclavian for arteriovenous aneurysms, no deaths.

RECAPITULATION

29 operations upon subclavians, arterial.
7 operations upon subclavians, arteriovenous.

36 Total subclavian operations, with three deaths. Mortality, 3:36, or 8.2 per cent

* Ref. bibliography Nos. 8, 19, 62 and 78.

SURGERY OF AXILLARY VESSELS*

Axillary Surgery for Arterial Aneurysm:

1 axillary arterial aneurysm was cured by double ligation of the artery at the seat of injury. (In two others the third subclavian was ligated.)

Axillary Surgery for Arteriovenous Aneurysms:

3 axillary arteriovenous aneurysms were cured, two by transvenous suture and one by quadruple ligation with excision.

Axillary Surgery for Conditions other than Aneurysms:

4 injuries in the course of radical extirpation of malignant growths:
- 2 ligations.
- 1 circular suture.
- 1 lateral suture.

8 Total, no deaths

SURGERY FOR ANEURYSMS OF THE BRACHIAL AND ITS BRANCHES, INCLUDING ANEURYSMS AND WOUNDS OF THE HAND†

Brachial Aneurysms:

3 arterial, all cured:
- 1 intrasaccular suture.
- 2 obliterative endo-aneurysmorraphies.

2 arteriovenous, all cured:
- 1 transvenous suture.
- 1 quadruple ligation.

5 Total, no deaths

Note: One brachial, not operated upon, was spontaneously cured.

Radial Aneurysms:

3 arterial, all cured:
- 1 obliterative endo-aneurysmorraphy.
- 2 restorative endo-aneurysmorraphies.

3 Total, no deaths

Aneurysms and Wounds of the Hand:

1 arteriovenous cavernous angioma of the hand, cured by excision.

1 arteriovenous wound cured by occlusion with clamp.

2 Total, no deaths

SURGERY OF THE ABDOMINAL AORTA

1 pathogenic (nonsyphilitic) in white male, age 23, involving abdominal aorta in proximity to celiac axis. Treated by various methods including celiotomy and peritoneal isolation of sac, secondary wiring and electrolysis (Moore-Corradi method). Temporary improvement. Death from subperitoneal rupture 54 days after operation.‡

1 colored female, age 28, syphilitic, ruptured aneurysm of the bifurcation, including both common iliac arteries, ligation of abdominal aorta with double cotton tape ligatures. Death from fulminating pulmonary hemorrhage, one year, five months and nine days after the ligation; with aneurysm clinically and anatomically cured.¶

* Ref. bibliography Nos. 19, 41, and 44.

† Ref. bibliography Nos. 1, 5, 32, 52 and 55.

‡ For discussion ref. bibliography Nos. 7, 9, 19 and 29.

¶ Ref. bibliography Nos. 53, 62 and 104.

2 abdominal aortic aneurysms treated by exploratory celiotomy and Colt's wire wisps, all operative recoveries with seeming temporary improvement.*

1 exploratory celiotomy, aneurysm in upper aorta exposed, no safe place could be exposed to wire. Abdomen closed without wiring. Death 25 hours after operation from rupture of the sac and fulminating hemorrhage into left pleural cavity.*

2 exploratory celiotomies for suspected aortic aneurysms; no aneurysms found; other pathology corrected, patient cured.*

7 Total, no deaths attributable to operation

SURGERY OF THE EXTERNAL, COMMON AND INTERNAL ILIACS†

Arterial Aneurysms:

1 obliterative endo-aneurysmorraphy, aneurysm involving the common and external iliacs, cured.

2 bands to external iliac for aneurysms of the external iliac, both cured.

3 Total, no deaths

Note: A case of bilateral aneurysms of the common iliac was cured by ligation of the abdominal aorta, and is tabulated under abdominal aorta.

Arteriovenous Aneurysm:

1 transvenous suture for aneurysm of external iliac, died on third day after operation, from gangrene of a loop of ileum.

Conditions other than Aneurysms:

1 iliac ligation and resection for femoral tubercular adenitis. Gangrene of foot and leg on third day; amputation; death four weeks after operation, following gradual decline.

8 (4 bilateral) ligations of internal iliac as prophylactic hemostatic as first step of a Wertheim operation for malignancy of uterus, all operative recoveries.

13 Total, with two deaths. Mortality, 2:13, or 15.3 per cent

SURGERY FOR ILIOFEMORAL ANEURYSMS‡

Iliofemoral Arterial Aneurysms:

12 bands to external iliac (one relapse cured by obliterative endo-aneurysmorraphy), all cured.

1 obliterative endo-aneurysmorraphy (following relapse from band), cured.

1 ligation of external iliac, died on seventh day from surgical anemia and exhaustion.

14 Total, with one death

Iliofemoral Arteriovenous Aneurysms:

2 transvenous sutures, one cured, one died.

1 ligation of common iliac, attempted quadruple ligation, died.

1 quadruple ligation, cured.

4 Total, two deaths

18 Total iliofemoral aneurysms operated upon, with three deaths. Mortality, 3:18, or 16.6 per cent

* The details of these cases are given in the author's tables of individual records, previously referred to.

† Ref. bibliography Nos. 19 (special aneurysms), 28, 41, 49 and 83.

‡ Ref. bibliography Nos. 32, 34, 40, 44 and 45.

FEMORAL SURGERY*

Femoral Arterial Aneurysms:

9 obliterative endo-aneurysmorraphies, all cured.
1 reconstructive endo-aneurysmorraphy, cured.
2 ligations in wound, no sac, both cured.
2 intrasaccular ligations (in sac), both cured.

14 Total, no deaths

Femoral Arteriovenous Aneurysms:

9 restorative operations:
4 by detachment of anastomosis and preservation of both vessels by lateral angiorraphy, all cured.
1 by transvenous closure of fistula (transvenous arteriorraphy) with preservation of both artery and vein (in large varicose sac), cured.
4 by transvenous closure of fistula with preservation of artery and sacrifice of vein, all cured.
8 obliterative operations:
2 obliterative endo-aneurysmorraphies by suture of all openings in a common sac, both cured.
6 quadruple ligations, with division of the vessels at the anastomosis, all cured.
1 irregular emergency procedure—hemostasis by hemostats left *in situ*, patient died.

18 Total, one death, or 5.55 per cent mortality

32 operations for femoral aneurysms, with one death. Mortality, 1:32. or 3.1 per cent

FEMOROPOPLITEAL SURGERY

Femoropopliteal Arterial Aneurysms:

7 obliterative endo-aneurysmorraphies, all cured.
1 reconstructive endo-aneurysmorraphy, cured.
1 intrasaccular ligation (in sac)—died 24 hours following operation from preoperative anemia, shock and exhaustion, despite preliminary intravenous infusion and continued infusion during the operation.

9 Total, one death. Mortality, 1:9, or 11.1 per cent

Femoropopliteal Arteriovenous Aneurysms:

3 obliterative operations:
2 obliterative endo-aneurysmorraphies, by suture of all openings in a common sac, cured.
1 quadruple ligation, with division of the vessels at the anastomosis, cured.

3 Total, no deaths

12 femoropoplitical aneurysms operated upon, with one death. Mortality, 1:12, or 8.33 per cent

POPLITEAL SURGERY†

Popliteal Arterial Aneurysms:

37 obliterative endo-aneurysmorraphies, 35 cured, two died.
1 restorative endo-aneurysmorraphy, cured.
2 reconstructive endo-aneurysmorraphies, both cured.
2 cured by mechanical compression.

42 Total, two deaths. Mortality, 2:42, or 4.76 per cent

* Ref. bibliography Nos. 19, 32, 35, 41, 47 and 48.
† Ref. bibiliography Nos. 19, 32, 41, 47 and 48.

Popliteal Arterial Injuries:

2 amputations for popliteal injuries; gangrenous on admission. Recovery.

Popliteal Arteriovenous Aneurysms:

3 quadruple ligations, with partial excision of sac, all cured.

1 obliterative transvenous endo-aneurysmorraphy, cured.

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4 Total, no deaths

Conditions other than Aneurysms:

1 bivascular circular angiorraphy following excision of popliteal vessels for myxosarcoma, complicated by thrombosis at anastomosis; final recovery with amputation six months after bivascular anastomosis.

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49 Total operations upon popliteal vessels, with two deaths. Mortality, 2:49, or 4.0 per cent

TIBIAL AND PEDAL SURGERY*

Tibial and Pedal Arterial Aneurysms:

2 restorative endo-aneurysmorraphies.

1 tibial, cured.

1 dorsalis pedis, cured.

1 obliterative endo-aneurysmorraphy. High bivascular, tibial, cured.

1 reconstructive endo-aneurysmorraphy, tibial, cured.

Peroneal Arteriovenous Aneurysm:

1 restorative endo-aneurysmorraphy, cured.

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5 Total, no deaths

Note: There were no deaths and no gangrene in this group. Suture methods were employed in all; there were no ligations except in two other tibial wounds not included in the above cases—cases of fresh tibial wounds, both healing perfectly.

PART III

SUMMARY OF PROCEDURES EMPLOYED BY DOCTOR MATAS IN SIX HUNDRED AND TWENTY OPERATIONS UPON THE BLOOD VESSELS, FOR ALL CAUSES, FROM 1888–1940

Suture Methods:

68 obliterative endo-aneurysmorraphies (for aneurysms).

25 restorative endo-aneurysmorraphies (for aneurysms).

5 reconstructive endo-aneurysmorraphies (for aneurysms).

1 circular angiorraphy (popliteal), following excision for myxosarcoma.

2 sutures of axillary wounds (radical extirpation of malignancies).

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101 Total operations by suture methods, for all causes.

Occlusions with Bands, Ligatures, and other Methods:

89 bands (77 for aneurysms, 12 for conditions other than aneurysms).

402 ligations:

47 single ligations for aneurysms.

15 quadruple ligations for arteriovenous aneurysms.

340 for conditions other than aneurysms.

8 excisions (three for aneurysms, five for conditions other than aneurysms).

6 wirings for aneurysms.

3 exploratory celiotomies, for aneurysms, no wiring.

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* Ref. bibliography No. 52.

5 clampings for aneurysms and wounds.
1 aneurysmotomy and plugging orifices of wounded artery in vertebral canal for aneurysm.
1 intrasaccular ligation for aneurysm.
2 mechanical compressions (popliteal).
2 amputations for popliteal injuries, gangrenous on admission.

519 Total occlusion with bands, ligatures, and other methods for all causes

RECAPITULATION

101 operations by suture methods.
519 operations by methods of ligation (including aluminum bands).

620 Total operations

PROCEDURES EMPLOYED BY DOCTOR MATAS IN SIX HUNDRED AND TWENTY OPERATIONS UPON THE BLOOD VESSELS, FOR ALL CAUSES, FROM 1888–1940

SUTURE METHODS

68 Obliterative Endo-aneurysmorraphies (Matas), All for Aneurysms:
Arterial:
3 brachial, cured.
1 radial, cured.
1 external iliac and common iliac, cured.
1 iliofemoral, cured following relapse six months after band to external iliac.
11 femoral, cured.
7 femoropopliteal, all cured.
37 popliteal (10 high popliteal, all cured; 20 midpopliteal, 18 cured, two died; seven low popliteal, all cured).
1 tibial, high bivascular, cured.
Arteriovenous:
1 common carotid, cured with preservation of artery and sacrifice of vein.
1 left temporal branch of external carotid, cured.
2 femoral, all openings sutured in a common sac; both cured.
2 femoropopliteal, cured.

68 Total, with two deaths. Mortality, 2:68, or 2.9 per cent

30 Restorative Operations, All for Aneurysms:
5 reconstructive endo-aneurysmorraphies (all arterial)
1 femoral, cured.
1 femoropopliteal, cured.
2 popliteal, both cured.
1 tibial, cured.
10 restorative endo-aneurysmorraphies (Matas):
Arterial:
2 radial, cured.
1 popliteal, cured.
1 tibial, cured.
1 dorsalis pedis, cured.
Arteriovenous:
4 femoral, all cured. (Detachment of anastomosis and preservation of both vessels by lateral angiorraphy.)
1 peroneal, cured.
15 restorative endo-aneurysmorraphies (Matas-Bickham operation by transvenous suture):

2 common carotid; with preservation of artery and sacrifice of vein. One cured. One case, transvenous suture following relapse from band to common carotid; sudden death in one week of preexisting coronary disease.
1 left subclavian, cured; with preservation of artery and sacrifice of vein.
2 axillary, both cured; with preservation of artery and sacrifice of vein.
1 brachial, cured; with preservation of artery and sacrifice of vein.
1 external iliac; preservation of both artery and vein. Died from thrombosis of mesentery.
2 iliofemoral; preservation of both artery and vein. One cured; one died on eighth day of erysipelas and fatal septic, thrombophlebitic pyemia.
5 femoral, all cured; one with preservation of both artery and vein, and four with preservation of artery and sacrifice of vein.
1 popliteal, cured.

30 Total, with three deaths. Mortality, 3:30, or 10 per cent
1 bivascular circular angiorraphy (popliteal), following excision for myxosarcoma. Recovery.
2 sutures of axillary wounds (injuries in the course of radical extirpations of malignant growths). Recovery.

RECAPITULATION

SUTURE METHODS

68 endo-aneurysmorraphies, obliteratives; with two deaths
25 restorative endo-aneurysmorraphies; with three deaths.
5 reconstructive endo-aneurysmorraphies; all cured.
1 bivascular circular angiorraphy; with recovery.
2 sutures of axillary wounds; with recovery.

101 Total, with five deaths. Mortality, 5:101, or 4.9 per cent.

RECAPITULATION—ENDO-ANERYSMORRAPHIES

98 Total Endo-aneurysmorraphies:
68 obliterative; 68:98, or 69.22 per cent
25 restorative; 25:98, or 26.52 per cent
5 reconstructive; 5:98, or 5.10 per cent

98 Total endo-aneurysmorraphies, with five deaths.

Remarks: If we subtract three deaths due to extrinsic causes (one from coronary disease, one from thrombosis of mesentery, and one from erysipelas) we have a mortality of 2:98, or 2.4 per cent.

OCCLUSIONS WITH BANDS, LIGATURES, AND OTHER METHODS

89 Occlusions with Bands:
68 bands for arterial aneurysms:
20 proximal common carotid, with one death.
14 distal common carotid, with no operative deaths.
3 internal carotid, one death.
3 innominate, one death.
6 proximal subclavian, no deaths.
8 distal subclavian, no deaths.
14 external iliac and iliofemoral, no deaths.
9 bands for arteriovenous aneurysms:

8 common carotid, no deaths. (One death followed a second operation, transvenous suture, but was caused by preexisting coronary disease.)
1 subclavian, cured.
12 bands for conditions other than aneurysm, no deaths:
12 common carotid.

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89 Total band occlusions, with three deaths. Mortality, 3:89, or 3.3 per cent
402 Occlusions with Ligatures:
62 ligations for aneurysms:
Arterial (single ligatures—silk, catgut, tendon, cotton tape):
4 proximal common carotid, no deaths.
4 distal common carotid, no deaths.
1 internal carotid, cured.
3 external carotid (two meningea media), all cured.
2 provisional innominate ligations, no deaths.
1 permanent innominate ligation; with death from consecutive secondary hemorrhage and cerebral complications from infected gunshot wound of the carotid.
11 subclavian:
6 proximal, two right, four left, all cured.
2 third subclavian for axillary traumatic (pulsating) hematomata; one right, cured; one left, died of preoperative hemorrhage and shock.
3 distal, no deaths.
1 axillary, cured.
1 abdominal aorta, cured.
1 external iliac, died on seventh day from surgical anemia and exhaustion.
2 femoral ligations in wounds, no sac, cured.
Arteriovenous (single ligatures):
2 internal carotid, cured.
5 external carotid (three for cavernous angiomata of the scalp and ear), all cured.
4 provisional innominate, no deaths.
5 subclavian:
4 right, cured.
1 thyroid axis and branches, cured.
Arteriovenous (quadruple ligatures):
1 external carotid, cured.
1 axillary, cured.
1 brachial, cured.
2 iliofemoral; one cured; one attempted quadruple ligature, died same day of hemorrhage and shock.
6 femoral, all cured.
1 femoropopliteal, cured.
3 popliteal, with partial excision of sac, all cured.

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62 Total ligations for aneurysms, with four deaths. Mortality, 4:62, or 6.4 per cent
340 Ligations for Conditions other than Aneursym.
8 proximal ligations of the common carotid, mainly for malignant growths. Three deaths from cerebral complications.
90 ligations of the external carotid, mainly for malignant growths, with seven deaths (cerebral, three; pulmonary, three; cardiopulmonary, with a question mark, one).
34 ligations of branches separately ligated at time of external carotid ligation.

197 ligations of thyroid arteries, either as a preliminary to operation or in the course of operation for thyroid disease. (One death from laryngeal spasm.)

2 ligations of axillary arteries for injuries during the course of radical extirpation of malignant growths. Recovery.

1 iliac ligation for tuberculous adenitis; thrombotic occlusion; gangrene of leg; amputation; final death four weeks after ligation.

8 ligations of internal iliac in four bilateral ligations as a prophylactic hemostatic, as first step of a Wertheim operation for malignancy of uterus. No deaths.

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340 Total ligations for conditions other than aneurysms, twelve deaths. Mortality, 12:340, or 3.5 per cent

402 total ligations for all causes, with sixteen deaths. Mortality, 16:402, or 3.9 per cent

8 excisions or extirpations:

Arterial:

1 external carotid, excision of arterial varix, cured.

1 subclavian, excision of cervical rib for thrombotic occlusion and of blocked segment of left subclavian, cured.

Arteriovenous:

1 excision of cavernous (arteriovenous) metastatic angioma of chest, cured.

Conditions other than aneurysm:

5 excisions of common carotid with tumor; four recovered, one died.

6 Wiring:

Arterial aneurysms:

3 innominate-aortic, recovered from operation with variable periods of improvement and survival.

3 abdominal-aortic, recovered from operation with variable periods of improvement and survival.

3 exploratory celiotomies for abdominal aneurysms (no wiring):

2 cases, other pathology found and cured.

1 (no safe place to wire) patient died in 25 hours from rupture of aneurysm.

5 clamping:

Arterial wounds:

1 wound of inferior thyroid; emergency hemostasis by clamping subclavian; died third day from shock, surgical anemia and hypostatic pneumonia.

1 emergency hemostasis of internal mammary (with clamp) for stab wound causing fatal hemothorax.

1 intracranial control by forceps of middle cerebral for gunshot wound; death from preoperative hemorrhage and shock.

Arteriovenous wounds:

1 wound of palmar arch, cured by occlusion with clamp.

1 wound of femoral vessels, hemostats (clamps) left *in situ*; death.

1 aneurysmotomy and plugging orifices of bleeding artery in vertebral canal:

1 aneurysm of vertebral artery, cured.

1 intrasaccular ligation (in sac):

1 femoropopliteal. Died 24 hours following intrasaccular ligation from preoperative anemia, shock and exhaustion, despite preliminary intravenous infusion and continued infusion during the operation.

2 mechanical compression:

2 popliteal aneurysms cured by proximal mechanical compression.

2 amputations:

2 amputations for popliteal artery injuries; gangrenous on admission. Both recovered, with aneurysm cured.

RECAPITULATION

METHODS OF OCCLUSION, INCLUDING LIGATURE, BANDS AND CLAMPS

89 bands, three deaths.
402 ligations, 16 deaths.
8 excisions of aneurysms and segments of vessels, one death.
6 wirings for arterial aneurysms, no operative deaths.
3 exploratory celiotomies for aortic aneurysms, no operative deaths.
5 clampings, four deaths—emergency hemostasis of large arteries.
1 aneurysmotomy and plugging bleeding orifices of vertebral aneurysm, cured.
1 intrasaccular ligation, died. (Annandale procedure.)
2 mechanical compressions for aneurysm, cured. (Matas' calipers compressor.)
2 amputations for ruptured popliteal aneurysm—gangrene before operation; no deaths.

519 Total occlusions, with 25 deaths. Mortality 25:519, or 4.81 per cent
101 operations by methods of suture; five deaths. Mortality 5:101, or 4.9 per cent

GRAND TOTAL

620 operations, 30 deaths. Mortality 30:620, or 4.83 per cent.

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[49] 1924—Congenital Arteriovenous Fistula. Discussion of Dr. W. F. Rienhoff's paper. Meeting of A.M.A., June, 1924. (Manuscript received too late to appear in report of proceedings.)

[50] 1924—Subclavian Arteriovenous Aneurysm. Discussion of Doctor Reid's paper on personal experiences, quoted at meeting of A.M.A., June, 1924. (Unpublished.)

[51] 1924—Methods of Treating Aneurysms by Intrasaccular Suture (lantern slide lecture). Scientific exhibit, Motion Picture Theatre, A.M.A., Chicago, June 11, 1924.

[52] 1924—Observations de trois cas d'anévrismes Artériels Peripheriques de la Main et du Pied. Gueris par la Suture Intrasacculaire Conservatrice (endo-anévrismorrhaphie Reparatrice). Contribution to the "Livre d'or," in honor of Professor Emile Forgue, Montpellier, November 6, 1924. Reprint 8°, 1–20, illus. Masson et Cie, Paris.

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[54] 1924—Surgical Treatment of Chronic Valvular Diseases of the Heart. Discussion of Doctor Cutler's paper. Trans. Am. Surg. Assn., **42,** 175–179, 1924.

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Thirteenth Semi-annual Meeting, June 7, 1925. Demonstration of two cases of arteriovenous aneurysm. (Unpublished.)

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[60] 1923—Inaugural Presidential Address with Remarks on Endo-aneurysmorraphy. (Illustrated technic by moving films.) Read before the Clinical Congress of American College of Surgeons, Philadelphia, October 28, 1925. Surg., Gynec. and Obstet., **41**, 701–705, 1925.

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[62] 1926—Personal Experience in the Surgery of the Subclavian Vessels. Paper read at the meeting of the Southern Surgical Association at Biloxi, Miss., December 16, 1926. Trans. South. Surg. Assn., **39**, 213–227, 1926. Abstr., J.A.M.A., **88**, 595, 1927.

[63] 1927—Address at the Inaugural Meeting of the Chirurgical Society of Barcelona; with moving picture illustrating Dr. Matas' method of Endo-aneurysmorraphy for the Radical Cure of Aneurysm. Ars Medica, Barcelona, November, 1927 (in Spanish).

[64] 1927—Illustrated stereopticon lecture on the "Surgical Treatment of Aneurysms" (with special reference to Dr. Matas' method) before the Faculty and Student Body of the Medical School of Barcelona, October, 1927 (abstract in program, in Spanish).

[65] 1927—Tratamiento quirurgico de los Aneurismas por la Sutura Intrasacular. (Ponencias y Conferencias), Jornadas Medicas de Madrid, 18–23 Octubre, 1927, 536–545 (in volume of transactions, Madrid, 1928). Lecture, by invitation, on the "Radical Cure of Aneurysm"; illustrated by moving picture in the Assembly Hall of the University of Madrid during the Spanish Congress of Medicine, October, 1927 (see Trans. of the Congress, Madrid, 1928).

[66] 1929—Endo-aneurysmorraphy (Obliterative Type) for Radical Cure of Aneurysm. Remarks preliminary to a motion picture of the operation, as performed by the author at the Charity Hospital, on "Aneurysms of the Femoral, Popliteal and Tibial Arteries." Transactions seventeenth annual meeting Association of the Illinois Central Railroad System. Surgical Journal of Chicago, **34**, 215–218, 1929, and discussion.

[67] 1929—The Cause and Mechanism of Postoperative Embolism. Report presented at the Eighth International Congress of Surgery, Warsaw, July, 1929. (Rapports, 1, 137–144.)

[68] 1930—Postoperative Thrombosis and Embolism: The Present Status of the Question. One of the three scientific addresses delivered in honor of Dr. John Chalmers DaCosta on the "DaCosta Night" of the Philadelphia County Medical Society, April 30, 1930. Published in abstract in "The Roster," bulletin of the Society, April, 1930.

[69] 1930—Editorial in the American Journal of Surgery for July, 1930, on "Postoperative Thrombosis and the Contributions of the Scandinavian Surgeons to the Surgery of Embolectomy."

[70] 1930—Discussion of Professor Gynar Nystrom's paper on "Operative Relief of Pulmonary Embolism." Meeting of the American Surgical Association, Philadelphia, May 7, 1930. ANNALS OF SURGERY, **92**, 528–530, 1930.

[71] 1931—A discussion of Dr. A. Storck's paper on the "Treatment of Aortic Aneurysms

by the Method of Jugulo-Carotid Anastomosis (Babcock's Operation)." New Orleans M. and S. Jour., **84,** 448–454, 1931.

[72] 1931—On Autotransfusion. Letter to the New Orleans M. and S. Jour., **84,** 69–71, July, 1931 (establishing priority of W. S. Halsted, for this method of transfusion).

[73] 1932—Personal Experience in the Surgery of the Subclavians (illus. with lantern slides). Address at Scientific Session and Dedication Exercises of the Medical Department, University of Texas, Galveston, Texas, May 30, 1932. (No printed proceedings.)

[74] 1932—Personal Experiences in the Surgical Treatment of Aneurysms. Proceed. Am. Surg. Assn., New Haven, Conn., May 17, 1932; ANNALS OF SURGERY, **114,** 802–839, November, 1940.

[75] 1932—Discussion of Doctors Scott and Morton's paper on "The Treatment of Common Arterial Diseases of the Lower Extremities." J.A.M.A., **99,** 984–985, 1932. Meeting of A.M.A., New Orleans, May 13, 1932.

[76] 1932—The Donald C. Balfour Lecture, University of Toronto, April 5, 1932, on "Postoperative Thrombosis and Pulmonary Embolism, Before and After Lister: A Retrospect and Prospect." Bull. Med. Faculty, University of Toronto, 1932. Reprint 8°, 32.

[77] 1932—Cirsoid Aneurysm of the Face and Scalp. Synopsis of an illustrated report of a recent operation for the radical cure of a traumatic arteriovenous aneurysm (cirsoid) involving the left half of scalp and left parotid region. Four years' duration in a patient, age 24. Recovery, with cure of the aneurysm. Proc. South. Med. Assn. (Surg. Sect.) meeting held at Birmingham, Ala., November 15–18, 1932. South. Med. Jour., **26,** 820–826, 1933.

[78] 1932—Subclavian Arteriovenous and Arterial Aneurysms. Discussion of Dr. J. M. Mason's paper at the Miami meeting of the South. Surg. Assn., December 13–15, 1932. Am. Jour. Surg., **20,** 532–539, 1933.

[79] 1933—On the Indications and Methods of Surgical Treatment in Dealing with Aneurysms of Special Regions; as these have been evolved in the practice of the author. Lecture I, at the Post-graduate Assembly of the Fifth District Medical Society of Southern Texas. San Antonio, Texas, January 10, 1933. (No printed transactions.)

[80] 1933—A moving picture clinic of the "Methods of Treating Arterial and Arteriovenous Aneurysms in the Practice of the Author." Lecture II, at the Post-graduate Assembly of the Fifth District Medical Society of Southern Texas. San Antonio, Texas, January 12, 1933. (No printed transactions.)

[81] 1933—On the Use of Removable Aluminum Bands (Matas-Allen) in the Surgery of the Great Blood Vessels. A preliminary test of the efficiency of the circle of Willis in all operations involving the possible occlusion of the common and internal carotid; and other applications to the surgery of the subclavian, innominate and iliac arteries. A Round Table discussion, illustrated with lantern slides. Lecture III, Post-graduate Assembly of the Fifth District Medical Society of Southern Texas, San Antonio, Texas, January 13, 1933. (No printed transactions.)

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[89] 1935—Remarks (chiefly historical) on the Treatment of Peripheral Vascular Diseases by Alternate Positive and Negative Atmospheric Pressure by the Paevex Treatment of Reid and Herrmann. Discussion following paper on this subject, by Dr. Isidore Cohn in Orleans Parish Medical Society, March 11, 1935. New Orleans M. and S. Jour., **88,** 79–81, 1935.

[90] 1935—On Arteriography. Discussion following reading of Dr. J. Ross Veal's paper on "Recent Diagnostic and Therapeutic Advances in Peripheral Circulatory Disease." Trans. Louisiana State Med. Soc., New Orleans, April 29, 1935. New Orleans M. and S. Jour., **88,** 687-689, May, 1936. In this connection, see editorial New Orleans M. and S. Jour., **87,** 245–247, October, 1934. "The Matas Operation for Aneurysm in the Light of Arteriography."

[91] 1935—Contribution of Louisiana to the Surgery of the Blood Vessels. Address at the Centennial Celebration of the Foundation of the Medical School of Tulane University. Hutchinson Memorial, June 12, 1935. (Delayed printing for later publication in the author's History of Medicine in Louisiana, in advanced preparation.)

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[93] 1935—Contribution to the Casuistics of Cirsoid Aneurysms of the Scalp and Face: With Special Reference to their Surgical Treatment (illus.). In the testimonial volume dedicated to Professor Presno, of Havana. Revista de Medicina y Cirujia, Habana. Ano XL, No. 11, 869–884, November 30, 1935. (Printed in English.)

[94] 1935—On the Treatment of Carotid Cavernous Arteriovenous Aneurysms. Discussion following Dr. W. W. Dandy's paper on this subject. Trans. Am. Surg. Assn., **53,** 432–436, 1935. ANNALS OF SURGERY, **102,** 920–924, 1935.

[95] 1936—Femoral Aneurysms: Their Classification and Surgical Treatment (illus.). Lecture, Tulane Clinics, November 12, 1936. (Publication purposely delayed for additional data.)

[96] 1936—Discussion on "Aneurysms of the Extracranial Internal Carotid: Personal Statistics and Conclusions"—following paper by Drs. A. M. Shipley, N. Winslow, and W. W. Walker. Trans. South. Surg. Assn., **49,** 58–59, 1936; ANNALS OF SURGERY, **105,** 698–699, 1937.

[97] 1936—On Continued Intravenous Blood Drip of Marriott and Keknick. Discussion following reading of Doctor Silverman's paper on this subject. Orleans Parish Med. Soc., October 12, 1936. New Orleans M. and S. Jour., **89,** 545–548, 1937.

[98] 1937—Address on the Pioneers in the Transillumination of the Living Vascular

System for Clinical Purposes: With Special Reference to the Achievements of the Portuguese School of Vasoradiographers.

[99] 1937—Preamble to the citation and presentation of the Matas Medal to Professor Reynaldo dos Santos, of Lisbon, the second recipient of the Violet Hart Fund.

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[100] 1937—Discussion of Dr. Fred H. Krock's paper on "A Simplified Apparatus for Pressure-Suction Therapy for Vascular Disease." Trans. Surg. Section, So. Med. Assn., So. Med. Jour., **31**, No. 3, 294, March, 1938.

[101] 1937—Aneurysms of the Circle of Willis. Discussion of Doctor Dandy's "Intracranial 'Clip' Occlusion of the Internal Carotid for Aneurysms of the Circle of Willis," with supplementary remarks. Trans. So. Surg. Assn., Birmingham, December 7–9, 1937; ANNALS OF SURGERY, **107**, No. 5, 660–680, 1938.

[102] 1939—The Experience of the Charity Hospital of Louisiana, on Aneurysmal Diseases. (Dry clinic with lantern slides.) Thoughts on the "medical aneurysms" with special reference to the aortic and cerebral aneurysms. What is the medical and surgical outlook for the cure of these aneurysms? Clinic by invitation, at the Medical School of Louisiana State University, March 28, 1939. In connection with the visit of the American College of Physicians that date. (Unpublished.)

[103] 1939—Congenital Arteriovenous Angioma of the Arm; Metastases Eleven Years after Amputation. (Type of cavernous angioma histologically benign, clinically malignant) Trans. So. Surg. Assn., Augusta, Ga., December 3-7, 1939. ANNALS OF SURGERY, **111**, 1021–1045, June, 1940 (Illus.).

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[105] 1940—Discussion on "Experimental Studies in the Occlusion of Large Arteries," by Dr. Herman E. Pearse. Trans. Am. Surg. Assn., **58**, 1940. ANNALS OF SURGERY, **112**, 934–937, November, 1940.

[106] 1940—Final pictorial report on the case of Corinne Dunson, "Successful Ligation of the Abdominal Aorta for Aneurysm of the Bifurcation," to complete the preliminary report referred to in ref. 53 and 61 of this bibliography. Discussion and illustrations following papers on the successful abdominal aortic ligations of Doctors Elkin and Bigger. Trans. Am. Surg. Assn., **58**, 1940; ANNALS OF SURGERY, **112**, 907–908, November, 1940.

[107] 1940—Personal Experiences in Vascular Surgery. An historical sketch and statistical summary, covering the period 1888-1940. Trans. Am. Surg. Assn., **58**, 1940; ANNALS OF SURGERY, **112**, 802–839, November, 1940.

[108] 1940—Vascular Tumors. Piersol and Bartz's Cyclopedia of Medicine, Surgery and the Specialties. Vol. 15, 798-878, 2nd edition, revised, illustrated and augmented, 1940. Davis and Co., Philadelphia.

The foregoing summary has been prepared as an introduction to a series of articles by the author, to appear serially in the ANNALS OF SURGERY. These will include the "Surgery of Aneurysm: Its Regional Aspects," with the thought of eventually incorporating the essays into a monograph on the subject.

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CLINICAL AND EXPERIMENTAL OBSERVATIONS ON ARTERIOVENOUS FISTULAE*

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IN 1923, Lewis and Drury[1] presented observations on arteriovenous fistulae in man and in the experimental animal which were quite limited in scope, in that the clinical fistulae were apparently not operated upon, and the experiments dealt only with the immediate effects of a fistula in an animal which did not survive the experiment. Because of the obvious limitations of such observations, a number of the conclusions which were drawn up have since proved untenable. More specifically, cardiac output was said to be unchanged, whereas numerous observations[2, 3, 4] indicate that it is markedly increased and even doubled in the presence of a fistula. Fistulae were said not to affect venous pressure, although some of their own observations, and many observations by others, show a very definite increase and sometimes doubling of venous pressure proximal to a fistula, depending on the size of the fistula.

The dilatation of the heart was said to be due to the effect of deficient nutrition incident to a lowering of mean arterial pressures, whereas Green[5] showed an increased coronary flow due to systolic elevation, and others have demonstrated a degree of cardiac hypertrophy in dilated but properly functioning hearts totally incompatible with a deficient nourishment of cardiac muscle.

Since that time, a number of fistulae in man have been carefully studied before and after elimination of the fistulae, and numerous experimental animals have been observed before and after comparable fistulae were established between the larger vessels and permitted to remain for periods as long as seven years. In a number of instances, observations diametrically opposite to those of Lewis and Drury were made, and they are presented as substantiating the following concepts:

(1) That a fistula introduces a secondary or "fistulous" circuit into the circulatory system.

(2) That the lowered peripheral resistance in this secondary circuit results in a diversion of blood from the primary circuit with its high capillary resistance, the extent of such diversion and the consequent physiologic effects of such diversion depending upon the size of the fistula and its location in the arterial tree.

(3) That the diversion of blood from the normal arterial bed through the fistula into the capacious venous system proximal and distal to the fistula

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results in a lowering of blood pressure in the primary circuit, which may be fatal if the fistula is large, or if it lies between the larger vessels.

(4) That, following the production of a nonfatal fistula, the general blood pressure is lowered, the diastolic pressure permanently, but the systolic pressure is gradually restored to normal or even above the normal level by the compensatory phenomena of (a) an increased cardiac output through an acceleration of pulse rate in the presence of an increased venous filling; and (b) an increased total blood volume.

(5) That venous filling is increased as shown by an increase in venous pressure proximal to a fistula.

(6) That in a normally functioning, dilatable heart, this increased venous filling is easily disposed of by accelerated contractions and increased cardiac thrust without a rise in "general" venous pressure.

(7) That the part of the circulatory bed common to both the primary and the secondary circuits has an increased volume of blood flowing through it.

(8) That an "adjustment" dilatation of this part of the circulatory system occurs commensurate with the increased volume of blood flowing through it, the dilatation involving the artery and vein between the heart and the fistula, and all the chambers of the heart.

(9) That, when the fistula is first opened, this adjustment dilatation first involves only the thin-walled easily distensible, capacious venous bed, which, both proximal and distal to the fistula, receives blood under arterial pressure.

(10) That the immediate effect of the loss and diversion of blood from the primary arterial bed into the capacious venous bed is a decrease in the size of the heart, followed, in the nonfatal fistula, as the lowered blood pressure incident to this diversion is compensated by an increase in total volume of blood, by a gradual dilatation of the heart and of the artery proximal to the fistula, the final result being a dilatation of the entire circulatory bed through which the short-circuited blood flows.

(11) That closure of a fistula results in a reversal of all these changes, primarily as the result of the filling of the primary circuit by the blood formerly flowing through the secondary or fistulous circuit:

(a) An overdistention of an already dilated heart for 12–24 hours.
(b) A permanent elevation of diastolic pressure due to the elimination of an area of low peripheral resistance.
(c) A transient elevation of systolic and diastolic pressures for several days due to the distention of the arterial bed by the volume of blood increased in the presence of the fistula.
(d) Gradual reduction in the size of the heart and vessels to and from the previous site of fistula due to the reduction in the volume of blood flowing through that part of the circulatory bed common to both the primary and secondary circuits.

CLINICAL EXPERIENCES

Case 13*.—C. H., age 21, entered San Jose Hospital, March 31, 1932, six weeks

* Cases 1 to 12, inclusive, have been previously reported (see references 6 to 11).

after an accidental gunshot wound—the bullet of a .38-caliber pistol having entered the right thigh, laterally, coursing downward to lie under the skin on the inner aspect of the thigh just below Hunter's canal. Little difficulty had been experienced in controlling bleeding, and prompt healing of the wound occurred. About ten days after the injury, the patient first noticed a "purr" on placing his hand on the lower thigh, and in the days that followed, an expansile swelling gradually developed over this area.

Physical Examination.—When first seen by me, April 4, 1932, a continuous thrill and bruit were present over this swelling, most intense about six inches above the patella, directly in line with the femoral artery. Pressure over the site of maximum thrill successfully abolished the expansile pulsation in the large swelling, and no changes in pulse rate or blood pressure were noted. It was assumed, therefore, that the fistulous opening was small, and the patient was put to bed for complete rest to determine the possibility of spontaneous closure. During the week, definite improvement occurred, the swelling decreased, and the thrill and bruit seemed to diminish. No changes in blood pressure or pulse occurred on closure of the fistula by digital pressure, and, accordingly, operation was again deferred. At the end of the next week, the situation was exactly the same as at the preceding examinations, except for one fact: Closure of the fistula produced an appreciable increase in blood pressure from 118/55 to 124/64, and a drop in pulse rate from 88 to 80. This was considered conclusive evidence that a large fistula was present and that it probably would not close spontaneously.

Operation.—April 21, 1932: Spinal anesthesia. Because of the large pulsating swelling of the thigh, which was undoubtedly a false sac into which the fistula opened, the operation was performed under a tourniquet applied just below the groin. A long incision paralleling the femoral vessels was made in the midthigh. After displacing the sartorius muscle medially, a large false sac was entered and about a quart of organized and clotted blood was evacuated. The femoral artery was exposed and just above Hunter's canal, a rent 1.5 cm. long was found opening into the false sac. A similar rent was found in the companion vein, which also opened into the false sac, but the vein was in no way adherent to the artery. The artery and vein were ligated above and below the rents, and the vessels divided between the ligatures.

On removing the tourniquet, definite pulsation was noted in the stump of the ligated distal artery, indicating an adequate collateral circulation. At the end of the operation, the foot was warm and pink and a good pulse could be felt in the posterior tibial artery. An uneventful recovery followed.

Case 14.—W. B., age 38, entered Lane Hospital, March 7, 1933, three months following a gunshot wound of the right thigh. The bullet from a .22-caliber rifle entered the lateral thigh six inches above the knee, shattered the shaft of the femur, and lodged under the skin on the medial surface. He was taken to a hospital where the leg was placed in a Thomas splint. No nerve or vessel injury was suspected at that time. Three weeks later after sneezing, there was a sudden onset of throbbing pain in the lower thigh, which then began to swell to such proportions that the skin became tense and shiny. The pain was intense, a fever developed, but after a week both subsided. He was told that he had an arteriovenous fistula, but that operation would have to be deferred to await the development of collateral circulation. He left the hospital at his own request. Two weeks after leaving the hospital, a throbbing pain and fever again appeared, and he entered Lane Hospital for the surgical treatment of the fistula.

Physical Examination.—The heart was not enlarged; the sounds were normal; blood pressure, 100/66; pulse rate 84; the red cells 3,760,000; hemoglobin 61 per cent (Sahli); leukocytes 7,400. The right thigh was markedly swollen in the lower half of the medial and anteromedial surfaces. At the level of greatest swelling, the circumference of the right leg was 50.4 cm., left leg 37 cm. There was no visible pulsation, but a palpable one, and directly over the peak of the swelling there were a slight thrill and a continuous bruit, intensified in systole. The dorsalis pedis and posterior tibial pulses were absent on the right, normal on the left. There were no objective sensory changes, although the leg felt numb. The femoral artery on the right felt stronger than on the left, but com-

pression of the artery failed to produce any consistent alterations in the pulse or blood pressure. The continuous thrill and bruit, however, made the diagnosis of arteriovenous fistula a certainty, complicated by a large false aneurysmal sac.

Operation.—March 10, 1933: A tourniquet was applied just below the groin, and a long incision made from the medial aspect of the knee to a point halfway up the thigh, paralleling the course of the vessels. The muscles were separated in the direction of their fibers, and a large false sac was entered, evacuating a great amount of blood clot. At the bottom of the sac were the femoral vessels, each one with a large defect in its wall, approximately 2.5 cm. long, each rent opening separately into the false sac. Only a small segment of the wall of each vessel remained intact (Fig. 1).

The blood from the artery poured first into the false sac, and was then forced through the opening in the vein. The variable amount of blood flowing through the sac accounted for the variable effects of the fistula noted before operation. It seems probable that when the pressure in the sac became almost arterial, it tended to collapse the vein and

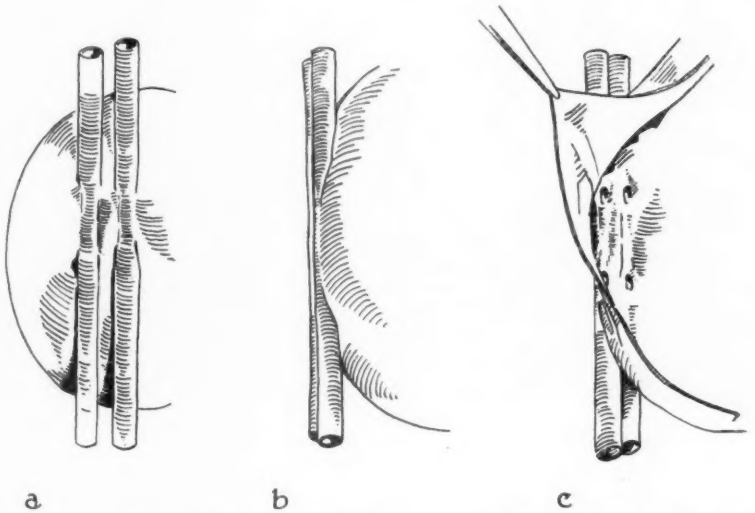


FIG. 1.—Case 14: Diagrammatic presentation of an unusual type of arteriovenous fistula of popliteal vessels: Proximal artery and vein both open into a large false sac, with separate openings into distal artery and vein. Quadruple ligation and excision performed. Restoration of vessels impossible. No impairment of circulation.

its opening, the flow of blood would cease temporarily, the continuous murmur would cease, and other attributes of a fistula would disappear. It was obviously impossible to restore the continuity of the artery, and, accordingly, a quadruple ligation was performed, the artery and vein divided between the ligatures, thus permitting their retraction. The tourniquet was removed and all bleeding points were controlled. A good pulsation was present in the stump of the distal artery, indicating an adequate collateral circulation. At the completion of the operation the color of the foot was excellent, although the posterior tibia and dorsalis pedis pulses were absent. An uneventful recovery followed. On dismissal the general blood pressure was 104/72.

Case 15.—C. S., age 23, entered Lane Hospital, October 17, 1933, complaining, mainly, of recurring ulcers of the left leg since a gunshot wound of the left thigh four years previously. The shot entered the left thigh just below Poupart's ligament, apparently grazed the femur, a fragment of the bullet being recovered from underneath the skin posteriorly. The initial marked bleeding was surprisingly easily controlled. Immediately after the injury, the distal part of the extremity increased greatly in size; within a short time prominent varicosities appeared over the entire left leg, and within a year, the first ulcer

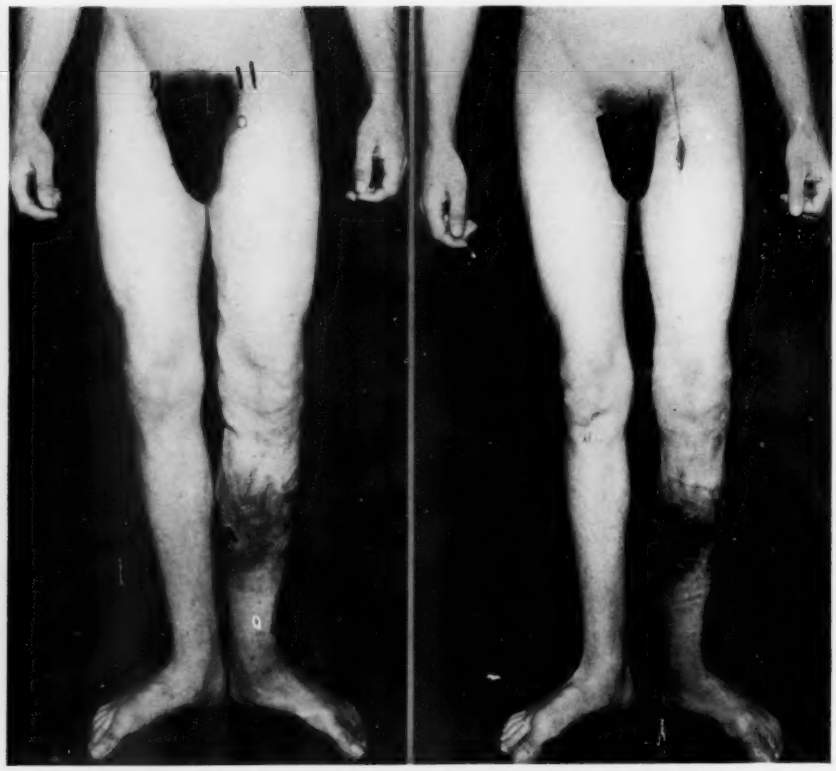


FIG. 2.—Case 15: Femoral fistula of four years' duration. Note marked dilatation of proximal artery, and great distention of all veins in the presence of the fistula, and their complete disappearance after operation.

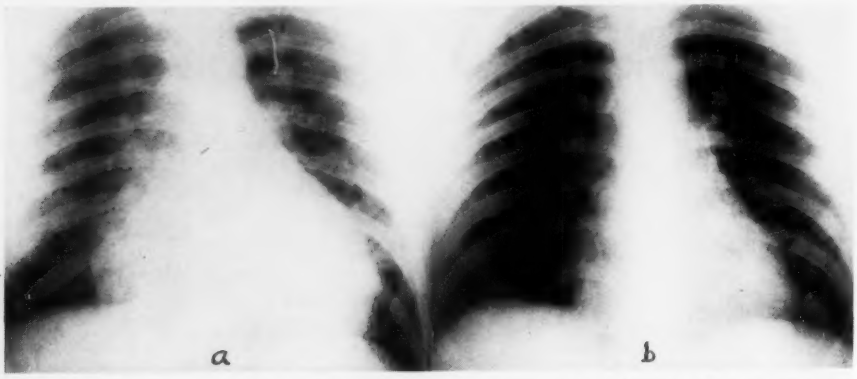


FIG. 3.—Case 15: Cardiac dilatation of marked degree accompanying a femoral fistula of four years' duration. Blood pressure markedly affected by closure of fistula. Total blood volume decreased 1,000 cc. and heart size markedly decreased within 30 days following elimination of fistula.

appeared on the lower leg followed by alternate healing and recurrence. A rumbling thrill at the site of injury was noted a few weeks after the accident and had been present ever since. There were no other complaints except a moderate thumping of his heart and beating in his ears, more pronounced after exercise, although it had not interfered with his work.

Physical Examination.—The left leg was markedly swollen in the dependent position (Fig. 2), visibly less in the horizontal position, and almost not at all with the leg elevated 45°. In this elevated position, definite visible pulsation was observed in the superficial veins of the thigh and leg down to the level of the ulcer on the anterior surface of the lower leg. There was marked pigmented discoloration of the skin around this ulcer and the leg, as a whole, was cyanotic as compared to the normal right leg. Just below Poupart's ligament was an area of visible pulsation about 3 cm. in diameter, into which ran a greatly dilated, palpable femoral artery. A very pronounced thrill and bruit were present over the area of visible pulsation, the bruit extending well up into the abdomen. The heart was greatly enlarged, the apex beat being in the sixth interspace at the anterior axillary line (Fig. 3 a). There was a soft systolic murmur at the apex. The radial pulse had a collapsing character, comparable with that of an aortic insufficiency. The posterior tibial and dorsalis pedis pulses were absent. On occluding the fistula by digital compression over the common femoral artery and vein, the whole lower leg became suffused with blood, and the varicosities became greatly distended.

TABLE I.—Case 15.

BEFORE OPERATION

DATE	R.B.C.	W.B.C.	Hb.	Blood Pressure		Pulse		Blood Volume	O_2 Content Venous Blood	
				Fistula Open	Fistula Closed	Open	Closed		L. Leg	R. Leg
10-17-33	6.3	7800	95							
10-18-33				140-55	160-100	72	56			
10-19-33	6.0		95	144-56	150-90		64	7200	18.6%	15.0%
10-20-33	5..9			148-54	170-100	84	48			
10-23-33			96		158-90					
10-24-33	6.1									
10-27-33	5.5		96							
10-28-33				124-60		88				

OPERATION FOR ELIMINATION OF LEFT FEMORAL FISTULA.

10-28-33 3 P.M.	6.3		118	144-94			72			
6 P.M.				156-104			80			
9 P.M.	6.5		117	158-106			78			
10-29-33	6.2		108	152-94			64			
10-30-33	6.2		107	164-92			74			
10-31-33	6.1		107	145-82			78			
11-1-33	5.4		104	138-72			76			
11-3-33	5.8		107	148-82			62			
11-4-33	5.3		99	128-72			60			
11-6-33	5.5		98	126-74			60			
11-8-33	5.2		96	124-70			78			
11-10-33	5.2		96	118-70			80			
11-13-33	4.9		90	116-70			64			
11-15-33	5.2		91	130-80			64			
11-27-33	5.0		95	118-76			60	6200		
8-29-35	4.8		84	120-75			76			

Skin temperatures as determined by a thermocouple were as follows:

	Right	Left
Forearm	32.0°C.	32.0°C.
Midcalf	31.0°C.	33.5°C.
Sole	27.5°C.	28.0°C.

The increased surface temperature of the left lower leg was consistent with the arterial pulsation in the superficial veins, indicating the presence of arterial blood close to the surface.

Specially taken roentgenograms of the left femur showed it to be 2 Mm. longer than the right.

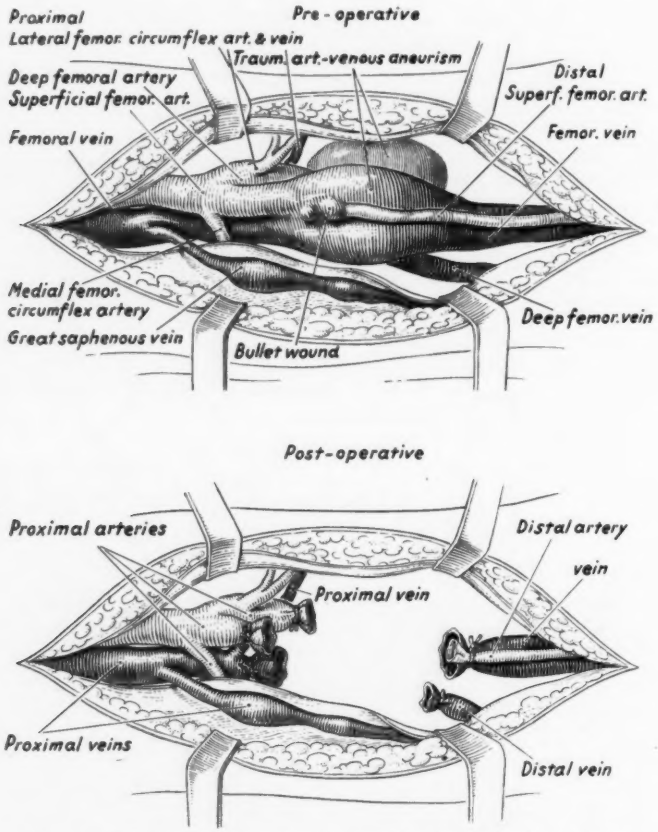


FIG. 4.—Case 15: Conditions found at operation for elimination of femoral fistula of four years' duration. Ligation of both deep femoral, and superficial femoral arteries was unaccompanied by impairment of nutrition.

During fluoroscopic examination, the heart was seen to increase 5 Mm. in diameter on closing the fistula. Special studies are recorded in Table I.

Operation.—October 28, 1933: Elimination of the fistula was performed without a tourniquet. A 20 cm.-long incision was made over the course of the femoral vessels. The large common femoral artery and vein were first exposed, isolated and surrounded with tapes which, however, were not tied. The enormously enlarged saphenous vein was displaced medially. The aneurysmal sac, about 3 cm. in diameter, was partially isolated, but a large extension of the sac down to the femur and into the soft tissues of the posterior

thigh was not isolated. It was found that both the superficial and deep femoral arteries entered directly into the aneurysmal sac, that the blood left the aneurysmal sac by passing into a small, almost obliterated superficial femoral artery, whereas the greater volume of the blood passed directly into a very large superficial femoral vein. It was apparent that the pulsation in the popliteal space which was attributed before operation to a large popliteal artery was in fact a pulsating popliteal vein. The vessels entering and leaving the sac were doubly ligated and the sac excised, except for the portion extending posteriorly (Fig. 4). The arteries ligated in this maneuver were: The superficial femoral artery just beyond the emergence of the deep femoral artery but proximal to the aneurysm; the superficial femoral artery beyond the aneurysmal sac; the deep femoral artery just distal to the lateral circumflex branch, but proximal to its entrance into the aneurysmal sac. No deep femoral artery was identifiable beyond the aneurysmal sac. The saphenous vein was spared. The superficial femoral vein was ligated proximally

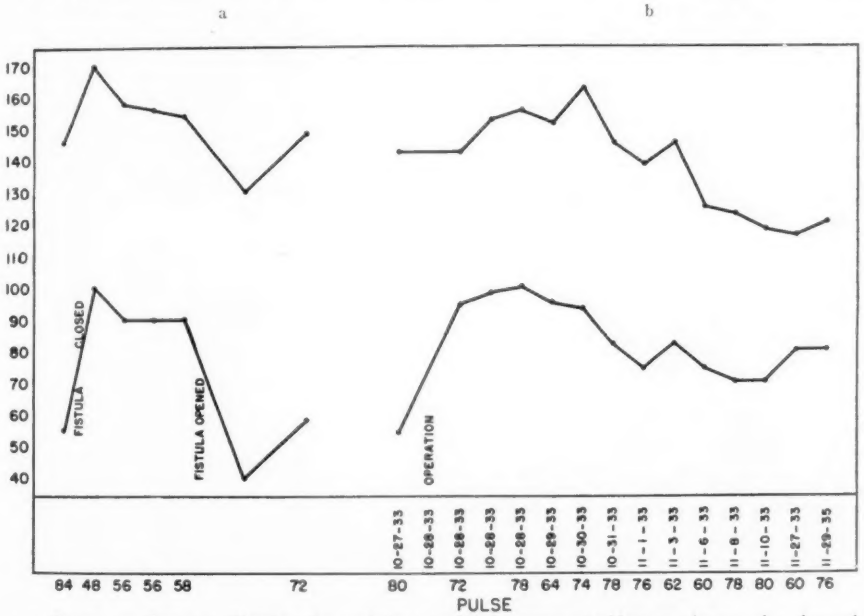


CHART 1.—Case 15: Showing the variations in blood pressure incident to closure of a femoral fistula of four years' duration: (a) By digital pressure; (b) by operative elimination. The blood volume decreased from 7,200 cc. to 6,200 cc. following operative removal of the fistula.

and distally to the aneurysmal sac, as were also several larger venous tributaries in the region of the deep femoral artery. The partially excised aneurysmal sac contained about two ounces of blood. The walls of that portion of the sac still remaining in the tissues were approximated with interrupted sutures. Although no pedal pulses were palpable, the foot was warm and pink at the end of the operation. At the beginning of the operation the blood pressure was 124/60, pulse rate 88. At the end of the five-hour operation it was 140/90, pulse rate 72. No fluids were administered either subcutaneously or intravenously during the operation.

The later behavior of the pulse and blood pressure are recorded in Chart 1. Blood studies (Table I) revealed a concentration of hemoglobin from 96 to 118 per cent (Sahli), and a concentration of red cells from 5.5 to 6.5 million in the first 24 hours after the operation.

In the course of the operation, a number of fragments of the original bullet were recovered, a few lying partially imbedded in the wall of the posterior extension of the aneurysmal sac. On the fourteenth day following the operation, the wound was reopened

and a large abscess evacuated, the pus coming from the blind aneurysmal sac in the posterior thigh. The wound was dakinized and an uneventful healing occurred. The heart gradually decreased in size, and four weeks after operation the total blood volume had dropped from 7,200 cc. to 6,200 cc. The pounding in his heart and head was no longer present after the operation. The ulcer of the lower leg rapidly epithelized, and was healed on discharge. Two years later, he returned for the injection of some prominent varicose veins of the left leg just above two small ulcers in the same area of previous ulceration. In other respects he was perfectly well.

Case 16.—S. H., age 22, entered Lane Hospital, March 8, 1937, because of an arteriovenous fistula of the left thigh, from which he had experienced no symptoms whatsoever, but which had been the cause for the rejection of a recent application for life insurance. He was told by his physician that unless the fistula were eliminated he would develop heart trouble.

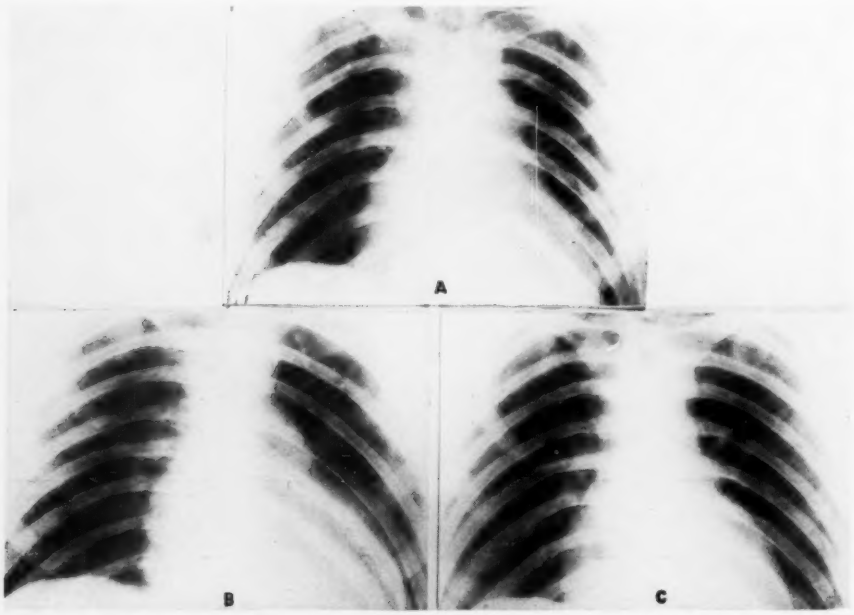


FIG. 5.—Case 16: (A) Cardiac dilatation in the presence of a femoral fistula of five years' duration, is (B) definitely increased 24 hours after the closure of the fistula, but (C) markedly decreased within 48 hours.

Five years previously, while on a camping trip, he had been shot accidentally through the left thigh by a .22-caliber pistol at a distance of about ten feet. The considerable bleeding which occurred was controlled immediately with a tourniquet, and there was no recurrence of bleeding. At the hospital, four hours later, a local débridement was performed, and he was discharged two weeks later with the wound healed, but with a "purr" in his thigh, which he himself discovered after his discharge, but which he did not call to his doctor's attention. There had been no cardiac symptoms, except an occasional light-headedness and a pounding in his heart noted only when lying down. He was a store clerk and had experienced no shortness of breath. His doctor noted a cardiac murmur for the first time about nine months before admission.

Physical Examination.—Temperature of 37.2° C., pulse rate 76, respirations 18. There was almost no swelling of the affected leg; even the superficial veins were not prominent. In the midthigh were a marked palpable thrill and audible murmur continuous throughout the cardiac cycle with great systolic accentuation. These were

maximum at a point midway between the pubic spine and internal condyle of the femur, but they could be felt and heard both distally and proximally to it, even over the lower abdomen.

There was marked increase in the precordial activity, and as the patient lay on his back one could see vigorous pulsation of the abdominal aorta, extending into the left inguinal region, continuing down the left femoral artery but ending abruptly in its midpoint at the presumed site of the fistula. On palpation, the left femoral artery appeared to have a diameter of approximately 9–10 Mm., whereas the right femoral artery felt about 5–6 Mm. in width. The popliteal, posterior tibial, and dorsalis pedis arteries could be easily felt on both sides. When the fistula was digitally compressed sufficiently to obliterate the thrill, the posterior tibial pulse below it was stronger than before, indicating a more than adequate collateral circulation. The heart was enlarged to percussion, measuring 3.5 cm. to the right and 10.8 cm. to the left of the midline (Fig. 5). A systolic

TABLE II.—Case 16.

BEFORE OPERATION

DATE	R. B. C.	W. B. C.	Hb.	Vital Capacity	Blood Volume	Venous Pressure Right Arm		Water Balance		Icterus Index	Pulse	Blood Pressure
						Fistula Open	Fistula Closed	Intake	Output Urine			
3-8-37	4.6	8600	86							88		
				4250		120	100	2500	1200		76	128-52
3-9-37				4300	5040	105	91	2500	800			
3-10-37						114		2500	1000			
3-11-37	4.8		99								80	138-58
3-12-37	4.5		100					2500	700			

OPERATION 3-13-37.

3-13-37									400			
2 hrs. PO	5.25		103					2500	2700		76	190-80
3-14-37	5.2		101									
	5.4		103								63	176-104
	5.4		104									
3-15-37	5.5		105					2500	1000	6.1	80	172-105
3-16-37	5.4		104					2500	900	6.3	74	176-100
3-17-37	4.9		98					2500	2000	4.4	76	140-70
3-18-37	5.0		100					2500	1950	5.8	72	130-90
3-19-37	4.8		98		4200			2500	650		84	142-88
3-21-37				4500								

murmur could be heard over the entire precordium, greatest at the apex but heard also over both the aortic and pulmonic areas. There was visible distention of the neck veins. The liver dulness was not increased, extending from the upper border of the sixth rib to a finger's breadth below the costal margin.

Studies of the effect upon blood pressure and pulse of closing and opening the fistula are recorded in Chart 2. On closing the fistula by digital compression, the blood pressure rose for a few seconds to 156/90, dropping to a level of 142/80 as long as the fistula was closed. Opening the fistula caused a precipitate drop to 118/48, recovering promptly to 138/56. The pulse rate was affected promptly by the closure of the fistula, dropping from 80 to 66 and 72.

The electrocardiogram was normal in all leads and was not altered by closure of the fistula, except for the drop in pulse rate.

Careful studies of the fundi revealed pulsating arteries and veins and arterioles in the region of a macula of normal size. When the fistula was closed, however, the arteries seemed to engorge and stop pulsating, the veins dilated and pulsated, and the arterioles of the macular region dilated and encroached more closely upon the macula.

Roentgenographic studies of the two femora demonstrated the left femur to be 1 cm. longer than the right. Inasmuch as the patient contracted the fistula at the age of 17, when the epiphyses were still ununited, we may ascribe this increased lengthening to the increased vascularity of the left thigh incident to the extensive collateral circulation, which occurs so characteristically in the presence of a fistula.

Fluoroscopically, "the heart is seen to be beating vigorously and at a rather rapid rate. When the fistula is compressed, the pulse rate slows, and the diameter increases 0.5 cm. As the compression of the fistula is continued the pulse rate is slightly accelerated, and the heart becomes even smaller than before closure of the fistula by 0.5 cm. On releasing the compression, the heart returns to its first state in about 30 seconds." These changes confirm previous observations that cardiac size varies with redistribution of the blood volume depending upon conditions at the fistula. The immediate effect of closing the fistula is to back up the previously short-circuited blood in the central arterial bed, particularly the heart, which temporarily dilates. There is a rise in arterial pressure which is promptly compensated by a peripheral dilatation (as seen in the retinal vessels). This filling of the peripheral bed by the blood formerly short-circuited through the fistula removes some of the blood volume in the central circulatory bed, and the heart becomes smaller than it is with the fistula open.

Operation.—March 13, 1937: The fistula was exposed by a long incision paralleling the femoral vessels in the midthigh. A tourniquet was not employed, as full and pulsating vessels are more easily identified and isolated than are collapsed ones. The proximal artery was freed first and a tape applied so as to be able to close it in case of trouble with bleeding. When the vessels and fistula were finally isolated the following measurements were obtained:

Proximal artery	1.0 cm. diameter
Proximal vein	2.0 cm. diameter
Distal artery	0.7 cm. diameter
Distal vein	1.2 cm. diameter

Quadruple ligation was performed and the fistulous area excised. By careful measurements the inside diameter of the fistula itself was computed as having had a diameter, in life, of 0.8 cm. The blood pressure at the beginning of the operation was 142/74, pulse rate 84. At the end of the two-hour operation it was 170/90, pulse rate 80. At the moment of ligation of the artery the blood pressure rose temporarily to 190/90, pulse rate 74.

Following operation significant changes occurred in the size of the cardiac silhouette (Fig. 5) in blood pressure (Chart 2), in pulse, and in the blood elements themselves, all changes being dependent upon the increase in total blood volume that occurred in the presence of the fistula, and that required several days for its readjustment to normal. Twenty-four hours after the elimination of this patient's fistula the transverse diameter of the heart had increased from a preoperative measurement of 19.5 cm. to 21 cm. Forty-eight hours after the operation the transverse diameter had decreased to 17.5 cm. The temporary postoperative increase in the size of the heart is explained on the basis of a redistribution of the total blood volume, and is the direct opposite of what occurs when the experimental fistula is first opened. Blood which formerly flowed freely through the fistula into a capacious venous system temporarily fills the arterial system, resulting in a temporarily increased general pressure and a temporary overdistention of the left cardiac chambers, possibly even extending back through the pulmonary vessels to distend the right cardiac chambers as well. Evidence of this is available from a study of roentgenograms made with the fistula open and with it closed. The roentgenologist's report was as follows: "Films were taken with ordinary exposure to show the normal lung

markings. The first film was taken without compression of the fistula and the second film was taken five seconds after the fistula had been shut off. When the two films were compared, we can see that the peribronchial markings are more prominent in the film taken after the fistula had been closed off. The heart measures about 5 Mm. more in transverse diameter in the film taken after closure of the fistula."

Following elimination of the fistula by operation the total blood volume decreases, the pressure slowly falls, and the heart decreases in size. The decrease in blood volume is effected in the first few days by elimination of the plasma resulting in a concentration of the cellular elements and in hemoglobin. As noted in the chart, the urinary output

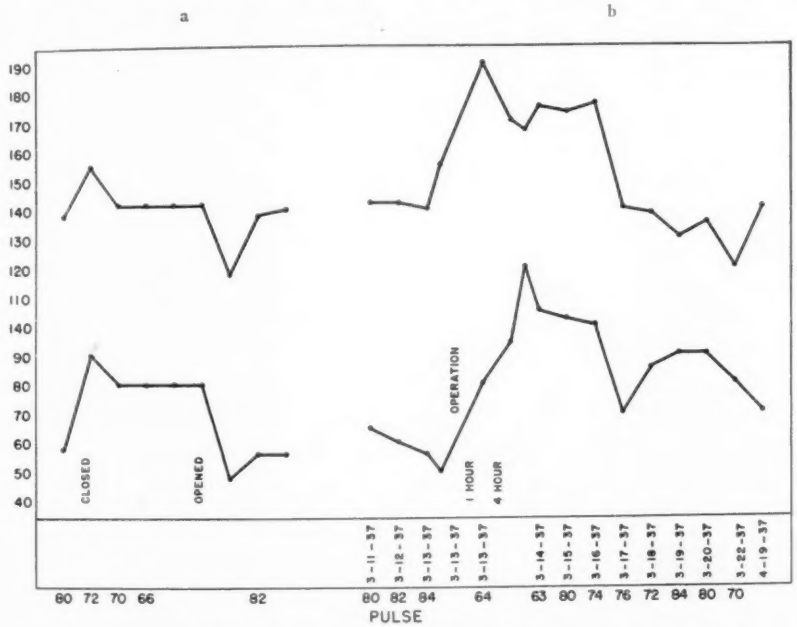


CHART 2.—Case 16: Variations in blood pressure incident to closure of a superficial femoral fistula of five years' duration: (a) by digital pressure; (b) by operation.
The blood volume decreased from 5,040 cc. to 4,200 cc. following elimination of the fistula.

greatly exceeded the intake of fluid during the first two days following the operation, suggesting a readjustment in total blood volume by the elimination of plasma. The red cells increased immediately after operation from 4.5 million per cc. to 5.4, dropping in five days to 4.8 million. The hemoglobin increased from 100 before operation to 105, dropping within five days to 98. The blood pressure and pulse rate behaved as noted in Chart 2. Immediately after operation, the blood which formerly leaked into the capacious venous system of the shorter circuit now filled the arterial system, as shown by an increased distention of the retinal arterioles. This overdistention of the arterial system resulted in a postoperative increase in blood pressure in the first few days as high as 190/80 and 172/105, as compared to 138/58 before operation. The overdistention of the aorta and cerebral vessels produced a vagal inhibitory effect upon the heart, causing a drop in pulse rate from 80 to 76 and 63. After six days, the blood pressure was 142/88 and pulse rate 84, the diastolic pressure being permanently elevated by the elimination of the lowered peripheral resistance due to the fistula. The total blood volume dropped from 5,000 cc. before operation to 4,200 cc. after operation.

Case 17.—J. H., age 38, entered Lane Hospital, April 3, 1939, on crutches, because of an injury to his left thigh one month previously. On March 4, 1939, while cutting linoleum with a knife, the blade accidentally entered the left midthigh to a depth of two

inches. On withdrawing the blade, blood spurted violently (about two yards) with each heart beat. Despite attempts at controlling the bleeding, he promptly fainted, apparently from loss of blood. He was taken to a local hospital where the wound was dressed and found to be filled with clot. About two hours after injury, the wound again broke open, with excessive bleeding, from which he again fainted. He left the hospital after two days, and uneventful healing occurred. The thigh became quite swollen, with black and blue discoloration, which gradually disappeared. He had not attempted weight-bearing because of pain on extension of the leg. A marked dyspnea, which was present for a few days after injury, had disappeared. About six days after the injury he noted a "buzzing" over the wound, which had persisted.

Physical Examination.—The heart was normal to percussion, but a loud blowing systolic murmur was heard at the apex. There was no gross difference in the two legs. On the midmedial aspect of the left thigh was a 2 cm. scar. Palpation over this area revealed a striking thrill and a small firm mass. On auscultation, a loud, continuous murmur could be heard, intensified in systole, and audible up and down the thigh along the course of the vessels. General blood pressure with the fistula open was 118/72, pulse rate 64. On compressing the femoral artery above the fistula, the blood pressure rose to 134/100, pulse rate 60. The left femoral artery was not demonstrably larger than the right. A good posterior tibial pulse could be felt on the left as well as on the right. The operation for the repair of the fistula was undertaken after only a month had elapsed since the injury because (1) healing had produced a painful thigh on motion; and (2) the changes in blood pressure and pulse rate on closing the fistula indicated a large fistula which, undoubtedly, would eventually have affected the heart, and would, most certainly, not have closed spontaneously.

Operation.—April 8, 1939: A tourniquet was not applied at the beginning of the operation. Under avertin and nitrous oxide anesthesia, Hunter's canal was exposed through a long, 15-cm. incision along the course of the vessels. The artery and vein were first isolated above the site of the fistula. An aneurysmal sac, 4 cm. in diameter, was identified, projecting from the vein, in which swirling arterial blood could be seen. The artery and vein distal to the sacculation were identified and isolated. On closing the artery proximal to the fistula with a clamp, pulsation was still present in the artery beyond the fistula (Henle-Coenen test), indicating an adequate collateral circulation. Despite this evidence, it was decided to ligate and excise the vein proximal and distal to the fistula in order to avoid the dangers of embolism of air or clot and to restore the artery. The vein was ligated above and below the fistulous opening and the intervening segment excised, together with a portion of the aneurysmal sac. The rent in the artery measured 1.2 cm. in length and was closed with fine, interrupted, oiled silk sutures, the line of suture being reinforced by suturing the distal stump of the vein so as to lie snugly over it. An excellent dorsalis pedis and posterior tibial pulse could be felt at the end of the operation. The blood pressure at the beginning of the operation was 104/70, pulse rate 80, and at the end of the three-hour operation it was 150/90, pulse rate 64. An uneventful recovery followed. No fluids were administered either subcutaneously or intravenously during the operation.

Case 18.—A. W., age 36, on September 19, 1932, accidentally sustained a gunshot wound, the bullet from a 32-40 deer rifle entering the right upper chest and emerging through the muscles at the base of the right neck. The furious bleeding which ensued was with difficulty controlled by digital pressure and dressings. No operation was performed, but the patient was apparently unconscious for about two hours. On the following morning, his local doctor noted a constant murmur in the right upper chest. The hand and arm felt numb for about a week, followed by a slow recovery except for a persisting numbness in the index finger. He returned to work exactly a month after the accident and continued his work as a clerk until 1937.

In April, 1933, he consulted a surgeon at the request of his local doctor. His com-

plaints at this time were paresthesia of the upper arm brought on by muscular movements of the arm, particularly abduction, numbness of the index finger, variable soreness of the right shoulder, and a booming sound in his right ear. Examination at this time showed a man apparently in robust health. His heart was of normal size (Fig. 6 a); there were no murmurs. Blood pressure was 140/80 in both arms, pulse 90. The scar of wound of entrance lay in the third interspace just to the right of the sternum, and

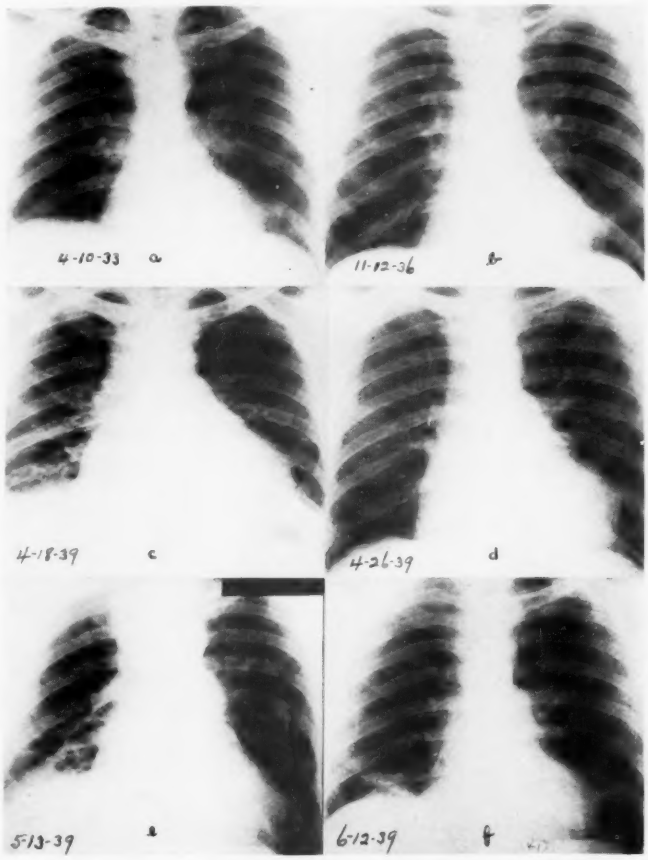


FIG. 6.—Case 18: Cardiac silhouettes in the presence of an arteriovenous fistula: (a) Six months after establishment of a right subclavian-jugular fistula. (b) Three and one-half years later—patient only slightly incapacitated. (c) Six and one-half years after injury when complete cardiac decompensation was present—generalized edema, ascites and hydrothorax. (d) Great improvement after one week's complete rest. (e) Three days after operation. (f) Thirty-three days after operation; all evidence of decompensation absent. The great vessels at the root of the heart are dilated, as well as the heart.

the scar of wound of exit lay on the ridge made by the trapezius muscle. There was no evidence of injury to the clavicle. In the right supraclavicular space was a slight swelling, and in this region one could hear a continuous bruit intensified in systole, which was modified but not obliterated by pressure just above the inner end of the right clavicle. Very deep and somewhat painful pressure at this point caused a fall in pulse rate from 84 to 76 per minute, and a very slight increase in blood pressure, from 138 to 144 systolic. There was slight atrophy of the muscles of the right forearm, but muscular movements and sensation were normal. In view of the apparent inaccessibility of the

lesion and because of its presumably innocuous character, the consulting surgeon advised against operation with the admonition to return for periodic observation. He was not seen again by this surgeon until September, 1936, when he returned complaining of increasing weakness, increasing shortness of breath, loss of weight, great nervousness due to the noises and "pounding" in his right chest, neck, and head, which were always worse in the night. Slight exertion produced great exhaustion, as did a heavy meal.

Physical Examination.—The patient was still in apparently good health, but his heart was larger than before (Fig. 6 b), and a faint systolic murmur could be made out

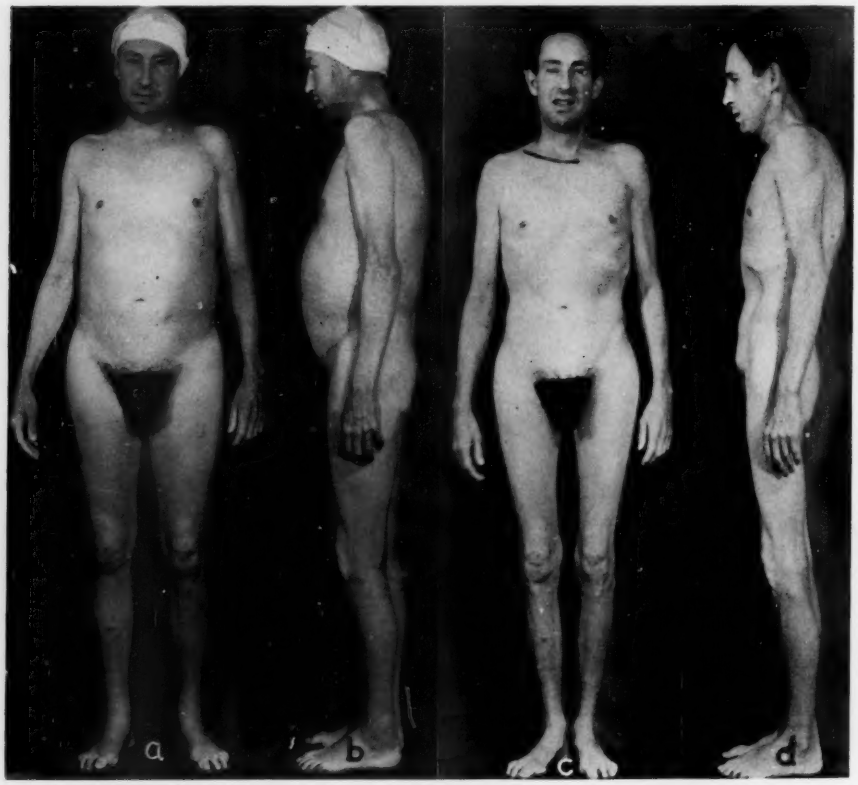


FIG. 7.—Case 18: (a, b) On admission, this patient showed complete cardiac decompensation, including severe brawny peripheral edema, ascites and hydrothorax due to right subclavian-jugular fistula of six and one-half years' duration. (c, d) Complete recovery, with loss of ascites and edema 12 days after elimination of fistula. Horner's syndrome, which was quite noticeable at this time, later practically disappeared. Line marks the incision.

at the apex. The other normal cardiac sounds were completely submerged by the tremendous roar emanating from the fistula. Deep pressure just above the inner end of the clavicle again modified the bruit but did not obliterate it. Blood pressure readings in the left arm were 125/55, right arm 122/55, pulse 90. On partially closing the fistula by painful digital pressure, the pulse dropped from 90 to 82 and the general blood pressure rose from 125/55 to 142/80. Despite the obvious increasing deleterious effects of the fistula, he again left the hospital without operation—the surgeon being impressed by the apparent impossibility of operating successfully upon a fistula lying presumably between the innominate vessels. In May, 1937, a right inguinal hernia was repaired under local anesthesia.

In April, 1939, he was referred to the Stanford Surgical Clinic. His story, since 1936, had been one of increasing breathlessness and easy fatigability, increasing dyspnea and palpitations on exertion, inability to work for two years, noctural dyspnea for two years, transient edema of the feet for two years, which finally became permanent four months ago when the edema extended upward to involve both thighs and, in the past two months, the abdomen as well. Dizzy spells and a persistent cough had also accompanied the increasing dyspnea. He had noted also a progressive weakness of the right arm and hand, which became quite purple and cold when all other extremities were pink and warm. When first seen on this admission he showed all the evidences of an advanced cardiac decompensation. He was markedly dyspneic, even while lying in bed. His face was cyanotic, the tissues everywhere slightly edematous, his lower extremities hard and indurated with edema, his abdomen prominent due to an advanced ascites (Fig. 7). There was marked venous distention of the cervical veins on the right, with visible pulsation in one small venous branch, and a vigorously pulsating carotid artery. A continuous thrill and bruit could be easily made out over the right upper chest and neck, most prominent just over the medial end of the clavicle. The heart was greatly enlarged with evidence of râles and increased dulness at both pleural bases. The urine showed no abnormalities. The plasma protein was 5.4 mg., the albumin 3.3 mg. per 100 cc. The following examinations were of more than usual interest (Table III):

TABLE III.—Case 18.

BEFORE OPERATION

Date	Weight in Kg.	W.B.C.	R.B.C.	Hb.	Pulse	Blood Pressure		Vital Capacity c.c.	Circulation Time in seconds		Venous Pressure cm. of water		Icterus Index	Blood Volume c.c.
						Right arm	Left arm		Right arm	Left arm	Right arm	Left arm		
4-8-33	77.2				90	140-80	140-80							
11-13-36	66				90	120-55	125-55							
4-18-39	79	10,260	5.25	105	120	142-86	130-78	1900	30	25	18	20.5	14	8080
4-24-39	62.5	7,200	5.55	112			118-64	2900	28	30	15	11		
4-27-39								3100					8	7700
5-2-39		10,400	5.1	92	82	118-60	122-64	3200	27	27	13.5	10.5		
5-9-39	62.2					116-58	116-58							7560

ELIMINATION OF FISTULA 5-10-39.

5-10-39			5.21 5.28	98 100	80		114-74							
5-11-39			4.8 4.97	91 97	80		140-60							
5-12-39					80		134-70							
5-13-39					84		132-80							
5-15-39					64		130-80						12	
5-16-39					78		134-84							
5-17-39					80		140-90							
5-18-39	58								30	17	20	8		6380
6-7-39	63				80	78-?	124-66	3300						5375
6-12-39									27	15	21	10		
6-19-39					88		104-68						8	
8-28-39	69	12,300	4.98	97	80	72-58	120-58	2800	15	10	10	10	2	

Complete rest in bed for eight days resulted in a remarkable recovery from the advanced cardiac decompensation on admission, as shown by the disappearance of his peripheral, pulmonary, and abdominal edema, a reduction of 17.4 Kg. in weight, a fall in general venous pressure, a fall in general blood pressure and pulse, a reduction in cardiac size and in the size of the liver, and a slight decrease in total blood volume.

Following three weeks' rest in bed, the patient was considered ready for operation. Medication during this time, in preparation for operation, had been as follows: A high caloric, high vitamin diet, limitation of fluids to 1,500 cc. daily, digitalis 0.6 Gm. daily for two days, teleostol vitamin capsules two daily, betaxin 10 mg. daily, and mercopurin 2 cc. intravenously on four occasions.

Operation.—May 10, 1939: Cyclopropane anesthesia. Because of the large blood volume and the possibility that closure of the fistula might result in an overdistention of the cardiac chambers with consequent failure, the left thigh was draped to expose the saphenous vein for venesection and for the withdrawal of blood if necessary. Evidence of such overdistention would have been a fall in blood pressure and increase in pulse rate on closure of the fistula instead of the usual increase in blood pressure and fall in pulse rate. The inaccessibility of the fistula had prevented ascertaining the exact effect of closing the fistula before operation. The incision was made in the folds of the skin from

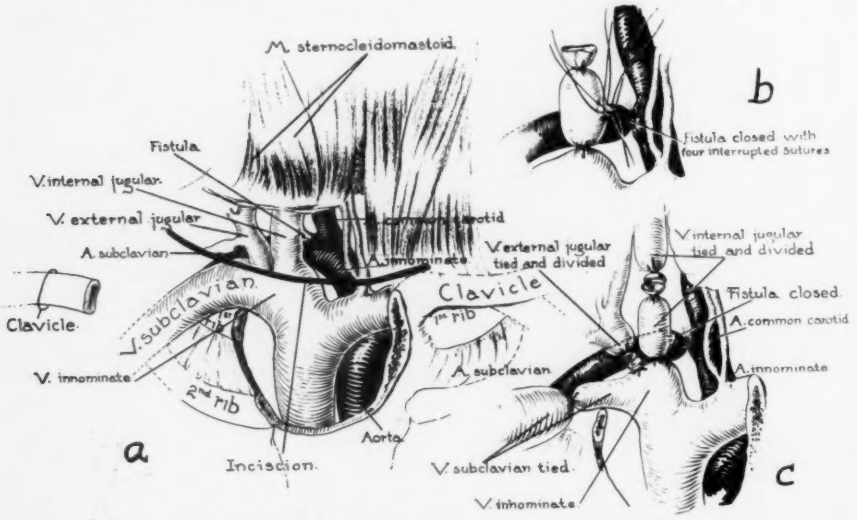


FIG. 8.—Case 18: Conditions disclosed at operation for subclavian-jugular fistula of six and one-half years' duration. Fistula eliminated by sutures in fistula itself, by ligation of subclavian artery in its first portion, by ligation of jugular vein distal and proximal to fistula, and by ligation of subclavian vein to balance the ligation of subclavian artery. No impairment of nutrition of the arm occurred.

the outer third of the clavicle to the left border of the sternum. A subperiosteal resection of the inner two-thirds of the clavicle was carried out. Scarring of the undersurface of the clavicle at the junction of the middle two-thirds indicated the site where the bullet had passed under the clavicle. The upper portion of the manubrium, together with the cartilaginous parts of the first and second ribs on the right, was removed. A hugely dilated innominate vein (Fig. 8) was identified and partially isolated, but the extreme thinning of its walls made its complete mobilization hazardous. Arterial blood was seen swirling vigorously through this vein. The innominate artery was isolated for tape control if necessary, followed by mobilization of the subclavian artery in its first portion, and in its third portion. The jugular vein was isolated, ligated and divided in the mid-portion of the neck. Reflecting this vein and mobilizing it down to its junction with the subclavian vein revealed the fistula as a short 5 Mm. wide tract running from the superior surface of the subclavian artery to the medial surface of the jugular vein (Fig. 8). It was thought that a ligature could be applied to the fistulous tract if properly isolated, but in attempting to free it, the fibrous wall of the fistula was torn, causing a rather alarming hemorrhage which was controlled by digital pressure. The subclavian artery was then permanently ligated in continuity in its first portion with braided silk, as was

the jugular vein at its entrance into the innominate. The rents in the subclavian artery and in the jugular vein were completely closed by interrupted sutures of silk. Because of the permanent ligation of the subclavian artery, it was thought necessary also to ligate the accompanying vein. Accordingly, the subclavian vein in its third portion was ligated in continuity. The thrill, bruit, and bleeding were thus all effectively controlled. The wound was closed in layers. Despite the six-hour operation, the patient at the end of it was in excellent condition, pulse rate 84, blood pressure 102/70; whereas at the beginning of the operation the pulse rate was 90 and the blood pressure 110/60. Immediately following ligation of the subclavian artery, the pulse rate dropped from 106 to 82 and

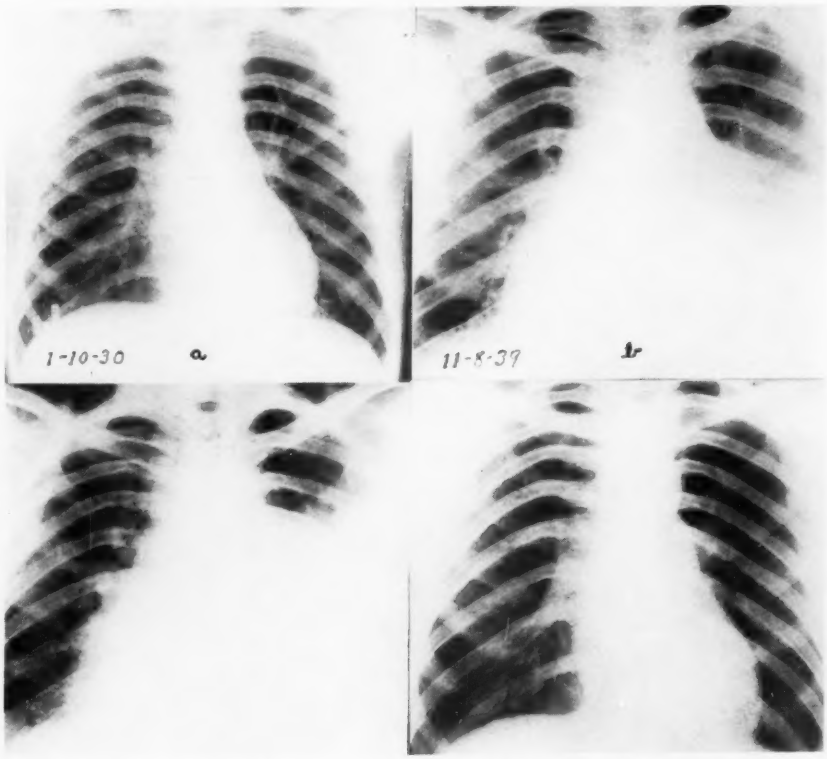


FIG. 9.—Case 19: Cardiac silhouettes in the presence of a popliteal fistula of ten years' duration: (a) At its inception. (b) In the presence of marked cardiac decompensation. (c) Following three weeks of complete rest there was no improvement. (d) Complete restoration to normal, two months following elimination of fistula.

the blood pressure rose from 90/60 to 120/74. No fluids were administered either during or after the operation, subcutaneously or intravenously. The closure of the fistula had the effect of providing a large autotransfusion of blood, thus accounting for the good condition of the patient.

One-half hour after his return to the ward a definite pulse could be felt in the right wrist, and at no time was there any apprehension about the circulation of the hand and arm. The blood pressure at this time was 114/74, pulse rate 80. There was a definite Horner's syndrome on the right, indicating that in the course of the operation the cervical sympathetics had either been severed or had been included in the ligatures. At 10 P.M., the hemoglobin had increased from a preoperative reading of 91 per cent to 100 per cent, and the red cells had increased from 5.1 million to 5.28 million, dropping again

the next morning to 93 per cent and 4.73 million. This temporary concentration of the blood elements is explained on the initial correction of the increased blood volume by an elimination of excess plasma. Two days after operation an icteric tint of the skin appeared, and his icterus index was 12, suggesting that the second correction of the increased blood volume was taking place, namely, a destruction of the red cells.

The patient rapidly recovered from his previous dyspnea and was discharged on the twelfth postoperative day, with instructions to remain in bed for two weeks while the previously hugely dilated heart recovered to normal size. He reentered the hospital, August 28, 1939, at which time he considered himself perfectly well. The Horner's syndrome was still present on the right. He had gained 12 pounds in weight since his operation in May. Other postoperative studies are recorded in Table III. The marked reduction in blood volume corroborated our experimental observations.

Case 19.—W. J., male, age 24, was admitted to St. Luke's Hospital, November 7, 1939, with a swelling of the left leg and abdomen, shortness of breath, and anorexia of one month's duration. A letter from the San Francisco Hospital stated that the patient had been admitted to that hospital, December 23, 1929, with a gunshot wound of the left thigh. At that time a diagnosis of arteriovenous fistula was made. A roentgenogram, January 10 (Fig. 9a), showed that "the heart shadows were within normal limits, and the lung fields were clear." He was discharged, January 25. He was readmitted to the same hospital, February 17, 1930, at which time a diagnosis of arteriovenous fistula was again made, but he was discharged to await the development of collateral circulation. His illness on admission to St. Luke's Hospital in November, 1939, was described as follows: One month previously the patient began to have sharp, sudden, stabbing pains in the left hypochondriac region, which were promptly followed by swelling of the ankles, legs, thighs, and abdomen. After two weeks in bed he attempted to get up, only to have the swelling reappear in his legs and abdomen. During the preceding three weeks he had been exceedingly short of breath until he was unable to sleep lying on his back. Only small amounts of food were eaten because of epigastric distress. Three weeks previously, he also began to be troubled with a deep, tight, unproductive cough. The patient stated that since the gunshot wound in 1929 he had led a very active life, participating in many sports.

Physical Examination.—Pulse 94, respirations 26, temperature 36° C., blood pressure 136/76. Chest showed absent breath sounds, dulness to percussion, and absent tactile fremitus over the entire left lower chest anteriorly and posteriorly. Breath sounds on the right were loud and hyperresonant to percussion, suggestive of a compensatory emphysema. The apex beat was 3 cm. outside the nipple line. There was a systolic thrill 2–3 cm. to left of nipple. Rhythm was irregular with a suggestion of gallop rhythm. There were no distinct murmurs. The abdomen was quite distended, with shifting dulness in the flanks (Fig. 10). He was tender throughout to deep palpation and particularly tender over the liver. Both legs showed extreme edema. There was a bullet scar on the medial surface of the left thigh 6 cm. above the knee, the wound of exit being 1 cm. lower on the posterolateral surface. There was a loud, coarse, continuous bruit heard over the entire left thigh, loudest immediately over the bullet scar on the medial surface. There was a distinct continuous thrill felt over this same area. Venous pulsations were observed in the lower leg and foot.

The patient stated that eight months previously he had been accidentally stabbed with a bayonet by a small boy, about 3 cm. above the left costal margin in the nipple line, the blade penetrating about 3 cm. Following this he had had pleurisy for a week with apparently no trouble thereafter.

On November 12, 1,420 cc. of yellow, clear fluid were removed from the left chest. In this fluid there were 625 white blood cells per cc. Revolta test was positive. Specific gravity was 1,013. This fluid was cultured and showed no growth. On November 28, 1,060 cc. of fluid were removed. On December 2, 250 cc. of bloody, turbid fluid were removed from the left chest. On December 11, 500 cc. of bloody fluid were removed. Following this

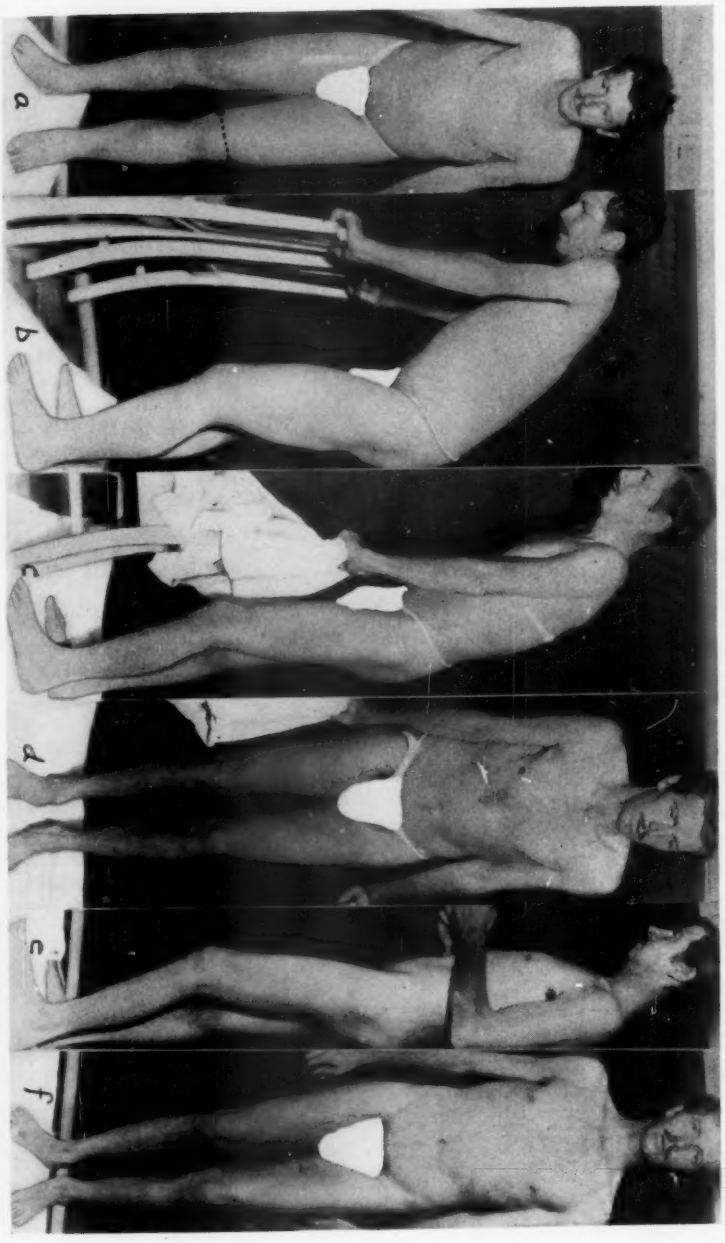


FIG. 10.—Case 19: (a, b) Marked evidences of cardiac decompensation accompanying popliteal fistula of ten years' duration. (c, d) Great improvement with complete rest in bed and frequent digital closure of artery proximal to fistula. (e, f) Complete restoration to normal following elimination of fistula.

last thoracentesis, the patient developed a high temperature. A pericardial friction rub could be heard throughout the cardiac cycle, and there was a pleural friction rub to be heard on inspiration in the third and fourth interspaces 2 cm. to the left of the sternum. On November 15, despite one week's rest in bed, there were still a large amount of fluid in the abdomen, marked edema of the right lower leg, and even more edema of the whole left leg.

On November 20 the following observations as to general blood pressure and pulse were made: Fistula open, B.P. 138/90, pulse 92; immediately after closing fistula, B.P. 190/106, pulse 72; during the continued compression of the fistula, B.P. 150/100; immediately on opening the fistula, B.P. 110/60; with prompt recovery to 138/76. From this date until time of operation the fistula was closed three and four times daily for periods of ten to 20 minutes with the hope of reducing the amount of blood flowing through the fistula and enabling the tissues around the fistula to contract. Following this date, salyrgan was administered three times—2 cc. on November 23; 1 cc. on November 26; and 1 cc. on November 29. There was prompt improvement in many respects.

On November 1, his red cells numbered 3,900,000, white cells 10,000, and hemoglobin was 84 per cent. On November 22, red cells numbered 4,100,000 and hemoglobin was 78 per cent. On November 25, his red cells had increased to 5,000,000 and hemoglobin was 83 per cent. On December 26, the day before operation, the red cells numbered 4,300,000 and hemoglobin was 81 per cent. On January 3, the red cells numbered 5,500,000 and hemoglobin was 87 per cent. On January 7, red cells numbered 5,000,000 and hemoglobin was 84 per cent.

The following studies in vital capacity were made: On December 12, it was 1,650 cc.; December 26, 2,620 cc. (54 per cent of normal); January 10, after closure of the fistula, 4,300 cc. (90 per cent of normal).

An electrocardiogram, November 10, showed the following: Pulse rate 100, p.r. interval 0.14 second, q.r.s. normal, amplitude and duration slight right axis deviation.

By December 10, a remarkable diminution in the peripheral edema had occurred; in fact, the left thigh looked slightly smaller than the right, indicative of slight muscular atrophy. Seven inches above the upper border of the patella, the right thigh measured 18½ inches, the left thigh 18 inches. Nine inches below the upper border of the patella, the right lower leg measured 12½ inches, the left lower leg 13½ inches. A good pulse could be felt in the posterior tibial and dorsalis pedis arteries on both sides. On closing the fistula by digital compression, the posterior tibial pulse disappeared on the left, but the color of the foot remained good. With the fistula open, pulse was 96, blood pressure 118/72. On closing the fistula, blood pressure was 134/90, pulse rate 76. The tremendous increase in blood pressure obtainable on November 20, on closing the fistula, was conspicuously absent. On palpation the right femoral artery had an apparent diameter just below the inguinal ligament of 5/16 inch; the left femoral artery measured 10/16 inch in diameter. On the day before operation, the blood pressure was 130/76, pulse 86, with the fistula open; with the fistula closed the blood pressure was 142/86, pulse 80.

Operation.—December 27, 1939: The patient was placed on his left side with the right thigh completely flexed on the abdomen. No tourniquet was used. A long incision was made paralleling the artery and the posterior border of the sartorius. The gracilis muscle was displaced posteriorly. The long saphenous nerve was mobilized and displaced posteriorly. The tendinous portion of the abductor magnus through which the artery passed was mobilized and divided in zigzag fashion. The artery and vein were hugely dilated down to the popliteal space. The fistula lay at the very apex of the popliteal space just 2 cm. beyond the emergence of a very large anastomonica magna (Fig. 11). The artery proximal to the fistula had a diameter of 1.8 cm., the vein proximal to the fistula was 2.2 cm. in diameter. Beyond the fistula the artery was 9 Mm. in diameter, the vein 2 cm. in diameter. Fortunately, it was possible to ligate the artery just beyond the anastomonica magna. A quadruple ligation was performed and the fistula excised. Following excision, a definite, though slight, pulsation could be seen in the stump of the

distal artery. The proximal artery was ligated twice, first with a large braided silk, and secondly with a transfixion suture of medium silk.

The following observations as to blood pressure and pulse were made during the operation: Fistula open, B.P. 115/60, pulse 88; proximal vein closed B.P. 130/70, pulse 90; proximal vein reopened, B.P. 118/62, pulse 100; proximal artery closed, B.P. 122/70, pulse 70; artery open, B.P. 108/56, pulse 100; proximal artery and vein closed, B.P. 134/72, pulse 80. Following excision of the fistula, B.P. 130/74, pulse 90.

Following the operation, the color of the left foot was good. The B.P. at 1:00 P.M.

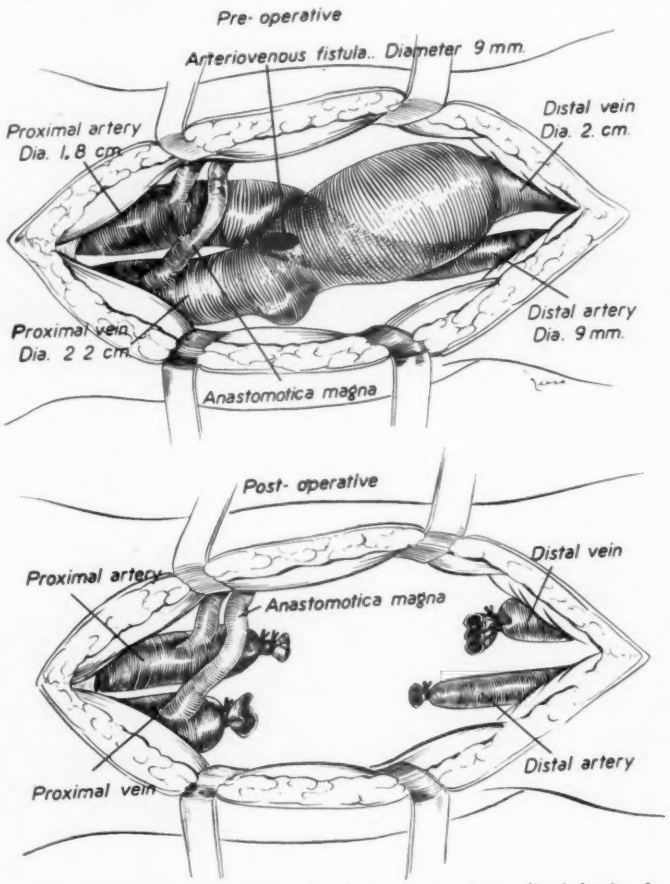


FIG. 11.—Case 19: Conditions found at operation for popliteal fistula of ten years' duration. Marked dilatation of proximal artery. Fistula eliminated by quadruple ligation and excision.

was 110/70; at 2:00 P.M., 122/82; at 3:00 P.M., 122/82; at 4:00 P.M., 112/70; at 5:00 P.M., 110/68; at 6:00 P.M., 124/84; at 8:00 P.M., 116/86. On December 28, B.P. 134/82, pulse 80; on January 1, B.P. 140/90, pulse 70; on January 3, B.P. 140/90, pulse 80; and on January 8, B.P. 134/96, pulse 80. On January 8 the apparent diameter of the left femoral artery was 9/16 inch, right femoral artery 6/16 inch. The circumference of the lower leg, nine inches below the upper border of the patella, was 12½ inches on the left and 12 inches on the right. The circumference of the thigh, seven inches above the upper border of the patella, was 16½ inches on the left and 16½ inches on the right.

The wound healed *per primam*. The patient was allowed out of bed, January 10, and was discharged from the hospital, January 14, 1940.

Case 20.—B. C., age 15, entered Stanford Hospital, June 14, 1939, for an arterio-

venous fistula of the left brachial vessels following an accidental gunshot wound ten weeks previously. The .22-caliber bullet entered the left upper arm two and one-half inches below the axillary fold on the medial surface, and emerged posteriorly, miraculously avoiding the bone. Severe bleeding occurred, but by the time the boy reached the hospital, one and one-half hours later, all external bleeding had ceased. The arm itself, however, promptly swelled to twice its normal size. As this swelling slowly disappeared, during the following four weeks, the patient noted a gradually increasing thrill over the site of the injury. He also noted an increased pounding of his heart, particularly when

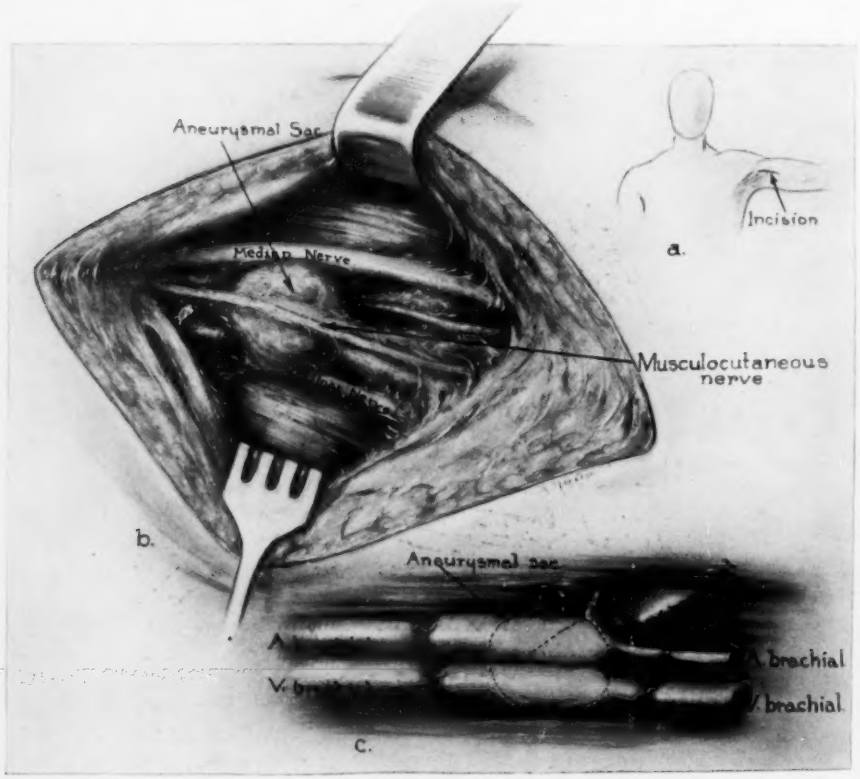


FIG. 12.—Case 20: Conditions at site of brachial fistula of ten weeks' duration. Quadruple ligation in continuity to avoid any possible injury to close-lying nerves.

he lay quietly in bed. There were no other symptoms. He had been able to play tennis without dyspnea.

Physical Examination.—The patient was a robust, healthy appearing young man. The left upper arm was slightly larger than the right, a loud bruit could be heard over the brachial vessels extending down toward the elbow, and upward as far as the base of the heart. There was no distention of veins. The heart was slightly enlarged, the apex beat being 2 cm. outside the nipple line in the fifth interspace. The thrill and bruit were easily stopped by pressure over the site of injury, the closure of the fistula being accompanied by a rise in blood pressure from 112/70, pulse 84, to 122/84, pulse 63. A preoperative total blood volume of 6,360 cc. was determined, which was approximately 600 cc. greater than would be predicted from his weight of 64.1 Kg. and his surface area of 1.74 sq. M. The consistent increase in blood pressure and fall in pulse rate on closing

the fistula were interpreted as indicating a large fistula that would not heal spontaneously, and operative elimination of the fistula was advised.

Operation.—June 16, 1939: Under gas anesthesia, a longitudinal incision was made over the brachial vessels. Considerable matting together of all structures, including the vessels themselves, the median, ulnar, and musculocutaneous nerves (Fig. 12a), made identification of structures difficult, but by working *without* a tourniquet the pulsating artery and full vein rendered the isolation of these structures easier. The musculocutaneous nerve lay flattened out immediately over a small aneurysmal swelling projecting from

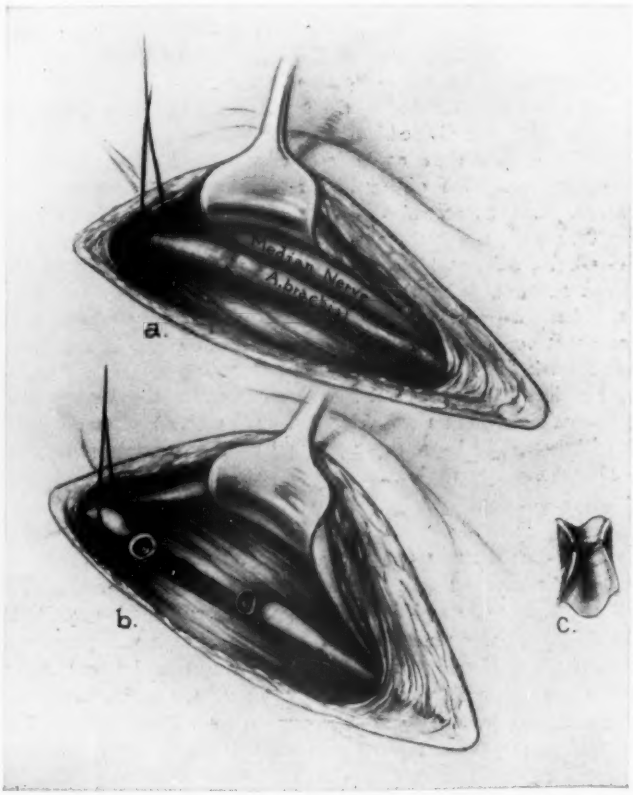


FIG. 13.—Case 20: At the second operation, following reactivation of the brachial fistula, it was found that the lumen of the brachial artery, proximal to the fistula, had been reestablished by the ligature cutting through the arterial wall, providing a lumen 3½ Mm. in diameter; the original artery having a diameter of 10 Mm. Simple excision of the previous ligature and ligation cured the fistula.

the vein. The proximal artery was dilated to 1 cm. in diameter, the distal artery having a diameter of only 3 Mm. Because of the matting together of the nerves and vessels, particularly in the region of the fistula, it was decided to perform a quadruple ligation of the vessels in continuity, and not to excise the fistula for fear of severing important nerve fibers. Following ligation of the four cardinal vessels with silk, all evidences of the fistula were absent. The wound was closed without drainage. At the operating table, just before closing the fistula, B.P. was 138/60, pulse 84. Following ligation of the vessels, B.P. rose to 144/80, pulse 76. The following day, B.P. 136/86, pulse 68. On the sixth postoperative day, B.P. 130/70, pulse 72. Healing *per primam* occurred, and the

patient was dismissed from the hospital on the tenth postoperative day, apparently cured of his fistula, and with a fair radial pulse. Six days following the operation a total blood volume of 6,000 cc. was found.

Exactly three weeks after the operation, he was again examined, and to our utter amazement (and chagrin) a thrill and bruit were again present. Pressure on the pulsating artery proximal to the site of the fistula caused the bruit to disappear. Localized pressure with a small pad over this vessel for several weeks did not affect the bruit. Accordingly, on August 22 the wound was reopened and to our surprise the silk ligature was found to have partially cut through the wall of the artery proximal to the fistula, reestablishing the lumen of the vessel and, thereby, reactivating the fistula (Fig. 13). The vessel was again ligated, and the segment containing the previous ligature excised. Complete and permanent disappearance of the thrill and bruit followed. The lesson is obvious: When performing quadruple ligation in those instances where excision of the fistula may not be feasible or desirable, the main proximal artery should be ligated not in continuity but ligated and divided.

Blood pressure studies were again instructive: On admission: 108/60, pulse, 76; two hours following religation of brachial artery: 124/70, pulse 70; first day postoperative: 124/74, pulse 82; second day postoperative: 130/76, pulse 70; third day postoperative: 130/80, pulse 70; fourth day postoperative: 120/66, pulse 80.

Case 21.—B. P., colored, male, age 10, was admitted to the San Diego General Hospital, August 13, 1939, 24 hours after an accidental gunshot wound of the left groin, the .22-caliber bullet entering at a point midway between the symphysis and the anterior superior spine about one inch above the inguinal ligament and emerging in the gluteal fold of the left buttock. Only a minimal amount of bleeding occurred. On examination, 24 hours after the accident, there was but little local swelling, the wounds of entrance and exit were small and closed, but a palpable thrill and audible bruit were most pronounced over the site of injury. The heart measured 11.7 cm. in transverse diameter. Red blood cells numbered 2,250,000, hemoglobin 67 per cent. He was discharged August 22, the thrill and bruit being still present.

On readmission, September 15, the heart had increased to 12.5 cm. in diameter; there was now a soft systolic murmur audible over the entire precordium, which was absent before. The red cells now numbered 3,330,000, hemoglobin 56 per cent. The thrill and bruit were, if anything, more audible than before. He was discharged, October 3.

On November 9, he was readmitted for operation, which was performed by me and Dr. Thomas O'Connell, November 15. Preoperative studies demonstrated a heart still enlarged, with the systolic murmur still present over the entire precordium. The red cells numbered 4,820,000, hemoglobin 88 per cent. A total blood volume of 2,875 cc. was determined. Blood pressure was 122/60, pulse 90. On closing the fistula by compression, the blood pressure increased to 130/80, pulse 80.

Operation.—November 15, 1939: A curved incision was made paralleling the inguinal ligament for about 10 cm. and then continuing down the inner aspect of the thigh for 6 cm. (in order to avoid crossing the creases of skin in the groin); the common femoral artery and vein were first isolated for temporary closure if necessary. As the dissection of these vessels was carried distally, it was found that the fistula lay between the common femoral artery just opposite the deep femoral branch and the superficial femoral vein. Its excision was accomplished (with considerable difficulty due to deep venous bleeding requiring several transfixion sutures) by ligation of the common femoral artery, the deep femoral artery, and the superficial femoral artery, and by ligation of the superficial femoral vein proximal and distal to the fistula, and the deep femoral vein. Before ligation, the common femoral artery measured 9 Mm. in diameter, the superficial femoral artery 4 Mm., and the deep femoral artery 2 Mm. In the excised state, the common femoral artery measured 4 Mm. in diameter, and the fistulous rent in the artery 4 Mm. in diameter. One may infer, therefore, that in the distended, or living state, the fistula had a diameter approximately equal to the size of the artery, or about 9 Mm.

Following excision of the fistula, the pulse dropped from 130 to 104. One hour after the operation, the pulse was 130, blood pressure 124/50. Twenty-four hours later the blood pressure was 118/82. A rapid diminution in the size of the heart occurred, its transverse diameter on November 24 being 10.6 cm., as compared to a preoperative diameter of 12.3 cm. A total blood volume determination showed a decrease of 300 cc. following removal of the fistula. The wound healed *per primam*, and at no time was there any doubt of the adequacy of the circulation of the extremity, which is rather extraordinary, considering the number of large vessels ligated. The reason, undoubtedly, lies in the stimulus to collateral circulation provided by the decreased peripheral resistance at the site of the fistula.

SUMMARY OF CLINICAL OBSERVATIONS

In analyzing these clinical experiences, the following points may be emphasized:

(1) Complete cardiac decompensation with peripheral edema, ascites, hydrothorax, and extreme dilatation of the heart may be completely corrected with return of the heart to normal size by the elimination of a peripheral fistula (Cases 18 and 19). This dilatation involves not only the heart but the great vessels at the base of the heart as well, as demonstrable by roentgenograms before and after elimination of the fistula (Cases 15, 16, 18, 19).

(2) Early evidence of the malign influence of a fistula upon the circulation—even before dilatation of the heart is detectable—is the behavior of the blood pressure and pulse following closure of the fistula by digital pressure: An increase in blood pressure and a fall in pulse rate, even though both are small, indicates that the fistula is large and is one which is not likely to close spontaneously, but is almost certain to produce increasing deleterious effects upon the circulation (Cases 13 and 17). In Case 18, failure to heed this evidence, first in 1933, when the heart was normal, and again in 1936, when the heart was beginning to dilate, led to a complete cardiac failure with great cardiac dilatation in 1939.

(3) This increase in blood pressure and fall in pulse rate is the first evidence that the circulatory bed through which the short-circuited blood flows is beginning to dilate, even though such dilatation may not yet be detectable by the usual means.

(4) The extent of the increase in blood pressure and fall in pulse rate depends upon the duration of the fistula, and is commensurate with the extent of the dilatation of the heart and vessels proximal to the fistula, being least in the fistulae of short duration with no or slight cardiac dilatation (Cases 13, 14, 17, 20 and 21), and greatest in the fistulae of long duration with great cardiac dilatation (Cases 15, 16, 18 and 19).

(5) A temporary but great increase in blood pressure and fall in pulse rate may occur immediately following the elimination of a fistula by operation, provided the operation is performed as a physiologic experiment without loss of blood (Cases 15 and 16). Despite prolonged operations, 4–6 hours long, the blood pressure at the end of the operation was higher and the pulse slower than at the beginning, due to an auto-transfusion from circulating blood which had increased in volume during the presence of the fistula (Cases 15, 16, 17, 18, 20).

(6) This temporarily great increase in blood pressure and fall in pulse rate on closing a fistula are dependent upon an increase in total blood volume, which is an inevitable accompaniment of a fistula of large size and long duration. In Case 15 the blood volume dropped from 7,200 to 6,200 cc. after the removal of the fistula, and in Case 16, the blood volume dropped from 5,000 cc. to 4,200 cc. after elimination of the fistula. Both cases showed marked cardiac dilatation and marked effects upon blood pressure and pulse, upon closing the fistula. A less marked effect on blood pressure and pulse occurred

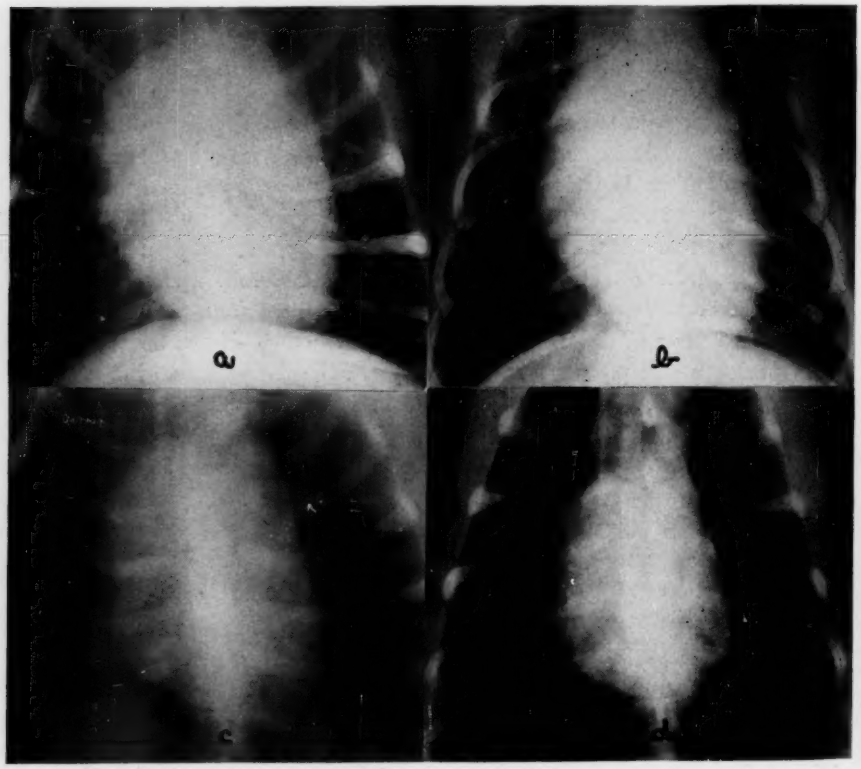


FIG. 14.—Animal 8: Roentgenograms of heart following establishment of an aorta-vena cava fistula: (a) 10:45 A.M., just before opening the fistula. (b) 11:15 A.M., 15 minutes after opening fistula. (c) 1:30 P.M. (d) 4:00 P.M. At 6 P.M. the animal died as the result of the fistula.

in Cases 20 and 21, in which the blood volume was reduced only 300 cc. in each case following elimination of fistulae of short duration. In case 18, blood volume was reduced from 8080 cc. to 7560 cc. by correction of the cardiac decompensation and from 7560 cc. to 5375 cc. by elimination of the fistula.

(7) The increased blood volume is reduced immediately following operative removal of a fistula by a reduction in the plasma as shown by increased urinary output (Case 16), and by a concentration of the red cells and hemoglobin in the blood (Cases 15, 16, 17 and 19). In Case 21, a marked dilution of blood followed the establishment of the fistula which was gradually corrected to practical normal figures.

(8) This increased blood volume may result in a transient overdistention of an already dilated heart following closure of a fistula by operation, due to a redistribution of the circulating blood, the volume of blood formerly diverted through the fistula into the capacious venous system now filling the central arterial bed (Cases 15 and 16).

(9) Eight cases of peripheral fistulae were eliminated by excision or ligature of segments of the main vessel to a limb without any evident effect upon the viability of the tissues beyond the ligature. In Case 21, the common femoral, deep femoral, and superficial femoral arteries were all ligated

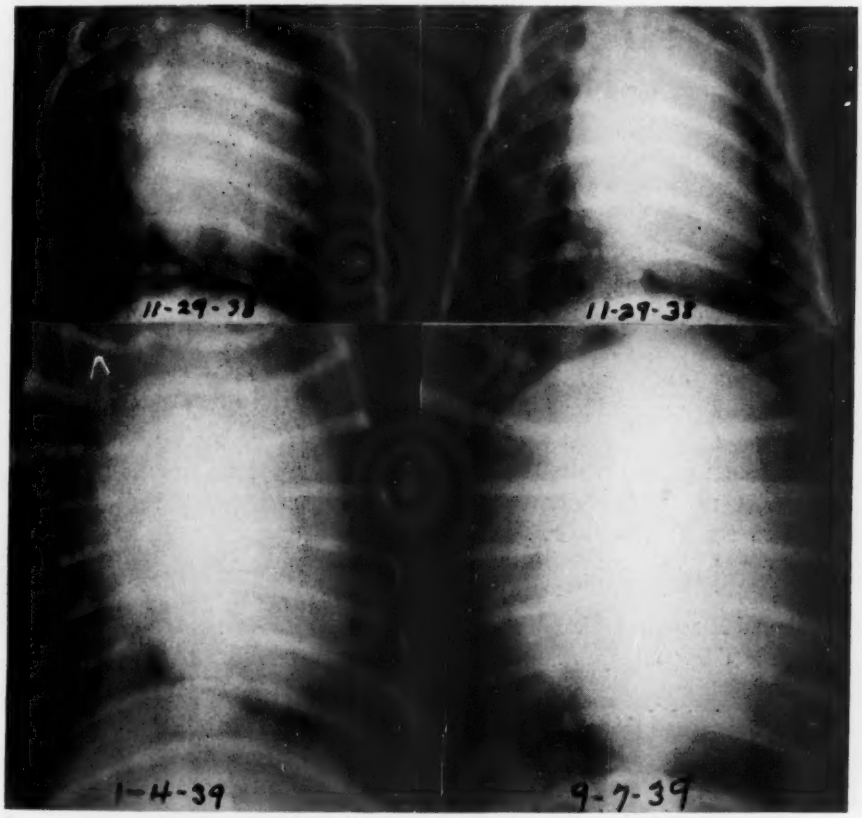


FIG. 15.—Pup 1: Variations in cardiac silhouette in animal that survives production of an aorta-vena cava fistula—an initial decrease in size is quickly supplanted by dilatation.

without impairment of nutrition or function of the leg. This is explicable on the basis of the stimulus to collateral circulation provided by the area of diminished peripheral resistance at the site of the fistula, which attracts blood to it through all available channels.

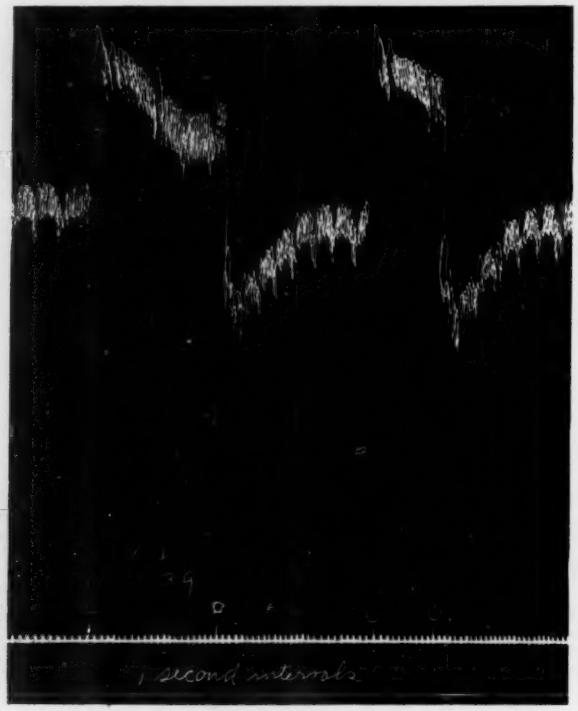
(10) When quadruple ligation of the vessels proximal and distal to the fistula is indicated, it would be desirable to ligate and divide the artery proximal to the fistula rather than to ligate it in continuity. In Case 20, the fistula was reactivated by the ligature cutting through the arterial wall and thereby reestablishing the lumen of the artery.

EXPERIMENTAL OBSERVATIONS

Animal 8.—Female, adult dog, weight 9 Kg. An aorta-vena cava fistula was established, January 24, 1939: aorta 6.5 Mm. in diameter, vena cava 8.5 Mm. in diameter, fistula 5.5 Mm. long. Before opening the fistula (10:45 A.M.), a roentgenogram of the chest was taken—pulse 120. At 11:15 A.M. a second roentgenogram showed a slight diminution in cardiac size—pulse 120. At 1:30 P.M. the cardiac size was even smaller—pulse 132—and at 4:00 P.M. the heart was remarkably smaller—pulse 200 (Fig. 14). At this time there was marked edema of both lower extremities. The animal died at 6 P.M., two hours later.

Pup 1.—Male, weight 9 Kg. On November 29, 1938, an aorta-vena cava fistula was established: aorta 6 Mm. in diameter, vena cava 8 Mm. in diameter, fistula 5 Mm. long. The pulse rate was immediately accelerated from 120 to 172 on opening the fistula.

According to roentgenograms (Fig. 15), a slight diminution in the cardiac shadow occurred immediately after the opening of the fistula, but within 24 hours the heart had recovered its original size, followed in the succeeding months by a truly enormous enlargement of the heart. At all times after the establishment of the fistula, good femoral pulsations were present on both sides. On April



GRAPH 1.—Pup 1: Kymographic record showing marked elevation of blood pressure and reduction in pulse rate on closing an aorta-vena cava fistula of 11 months' duration. Blood volume in this pup was 1,550 cc. as compared to 960 cc. in litter mate control.

24, 1939, the pulse rate was 192, respirations 52, the heart was very much enlarged and there was great dyspnea on exertion. On November 7, 1939, the animal was in much better condition, there was no edema, no ascites, good femoral pulsations were palpable, large veins were visible over the abdominal wall, and a loud bruit was audible over the lower abdomen. On this date, approximately one year after the establishment of the fistula, the carotid artery was cannulized to obtain a continuous record of the blood pressure. The fistula was exposed and closed by compression. Immediately, there was a most pronounced increase in blood pressure and a fall in pulse rate (Graph 1). Analyzing this kymographic record, we find that closure of the fistula increased the mean systolic pressure from 186 to 236 Mm.Hg. immediately after closing the fistula; dropping to a level of 212 Mm.Hg. as long as the fistula remained closed; falling precipitately on opening the fistula to 140 Mm.Hg., but recovering promptly within a few beats to the previous level of 186 Mm.Hg. Variations in pulse rate paralleled these changes in blood pressure. The animal was killed, and examination revealed an aorta dilated from the heart to the fistula and a greatly dilated vena cava from the fistula to the heart. The heart almost filled the chest. The fistula admitted a bougie having a circumference of

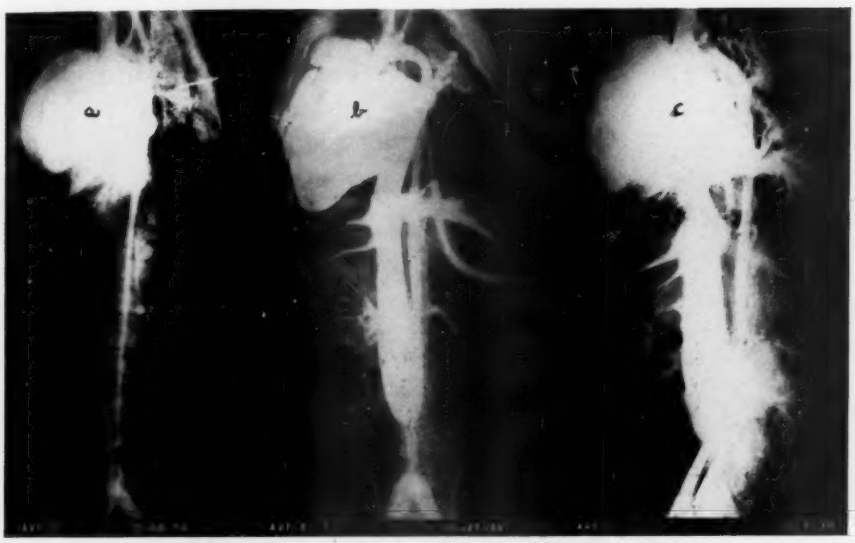


FIG. 16.—Roentgenograms of three litter mates following injection of circulatory system with bismuth oxychloride: (a) Normal heart, aorta, and vena cava of control animal. (b) Moderate dilatation of heart, aorta, and vena cava following establishment of aorta-vena cava fistula, which at death proved to be 12 Mm. in circumference. (c) Great dilatation of heart, aorta, and vena cava incident to fistula 18 Mm. in circumference. These injections prove that the dilatation involves the entire circulatory system through which the short-circuited blood passes, namely, the aorta from heart to fistula, the vena cava from fistula to heart, and all chambers of the heart.

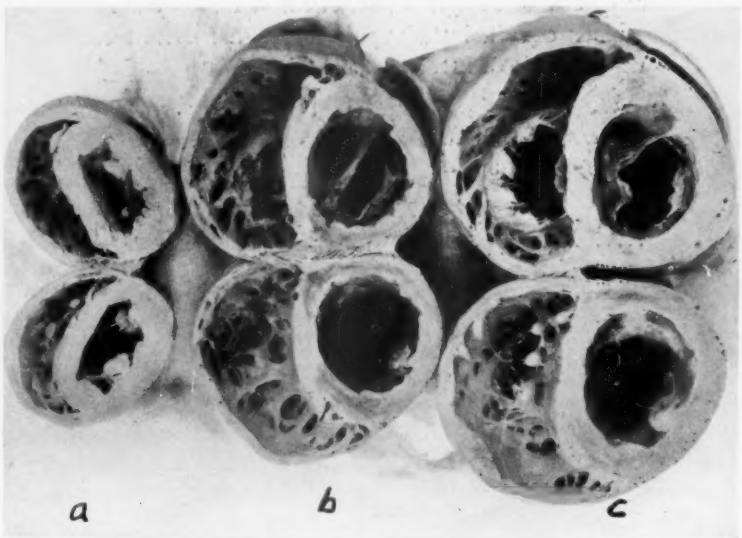


FIG. 17.—The dilatation of the heart is commensurate with the size of the fistula: (a) Heart of control dog; weight 74 Gm. (b) Heart of Pup 2 with an aorta-vena cava fistula 12 Mm. in circumference; weight 123 Gm. (c) Heart of Pup 1 with an aorta-vena cava fistula 18 Mm. in circumference; weight 176 Gm.
All animals from the same litter and approximately equal in size and weight.

18 Mm. The circulatory system was injected with bismuth oxychloride in gum acacia (Fig. 16). The heart weighed 176 Gm.; weight of animal 11.1 Kg. (Fig. 17).

Pup 2.—Male, weight 8.3 Kg. On December 6, 1938, an aorta-vena cava fistula was established: aorta 5 Mm. in diameter, vena cava 8 Mm. in diameter. On opening the fistula the pulse rate rose from 132 to 160. On the following day a good pulse could be palpated in both femoral arteries, there was no edema, and the pulse rate was 180. A roentgenogram taken 24 hours after establishing the fistula showed a definite decrease in the size of the heart. On December 8, 1938, the heart had recovered its previous size, there was a good femoral pulse to be felt in both groins, and the pulse rate was 200. The animal continued in good health until October 27, 1939. On this date, the heart had increased greatly in size, although not as much as the heart of the litter mate. The pulse rate was 140. A good femoral pulse could be felt on both sides, and there was no edema. The fistula was exposed and compressed, causing a fall in pulse rate from 140 to 104. The animal was killed and the circulatory system injected with bismuth oxychloride in gum acacia. The aorta and vena cava were both dilated from the fistula to the heart (Fig. 16), and the heart was greatly enlarged. The heart weighed 123 Gm., weight of animal 100 Kg. (Fig. 17). From the lesser cardiac enlargement as compared with Pup 1, it was considered probable that the fistula was smaller in this animal than in Pup 1. This proved to be the case. The fistula admitted a bougie having a circumference of 12 Mm., as compared with 18 Mm. in Pup 1.

The *control* animal was also killed on this date, and the heart and circulatory system injected with bismuth oxychloride (Fig. 16). The heart weighed 74 Gm., weight of the animal, 11.6 Kg. (Fig. 17).

The following observations on total blood volume in these three litter mates were made:

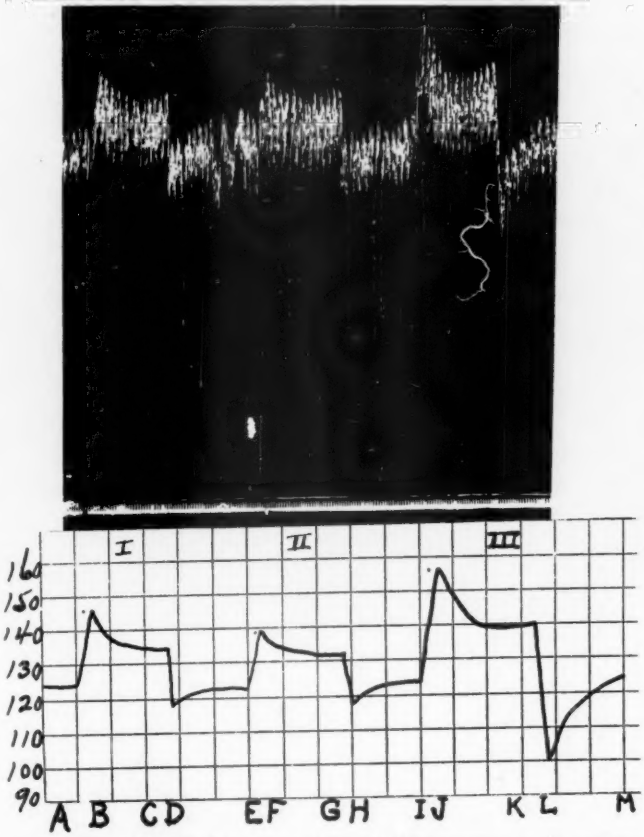
Animal	Dates	Weight	Blood Volume
Control	2/23/39	10.4 Kg.	950 cc.
	8/23/39	11.6 Kg.	960 cc.
Pup 2	2/28/39	10.5 Kg.	1,190 cc.
	9/20/39	10.7 Kg.	1,210 cc.
Pup 1	2/23/39	10.0 Kg.	1,430 cc.
	8/23/39	11.1 Kg.	1,550 cc.

By mathematical computation from measurements obtained in the roentgenograms of the injected animals (Fig. 16), the following approximate capacities were determined for that part of the circulatory system included in the shorter fistulous circuit, including the heart, the aorta from heart to fistula, and the vena cava from fistula to the heart:

	Capacity	Total Blood Volume	Increase in Capacity as Compared to Control	Increase in Total Blood Volume as Compared to Control
Control	200 cc.	960 cc.		
Pup 2	480 cc.	1,210 cc.	280 cc.	250 cc.
Pup 1	775 cc.	1,550 cc.	575 cc.	590 cc.

In these three litter mates, equal in weight and stature, there was a surprising correlation between the increase in capacity of the shorter circuit and the increase in the total blood volume, both being commensurate with the size of the fistula. It may be inferred that if no demonstrable dilatation of the heart or of the vessels proximal to the fistula occurs, there is probably also no demonstrable increase in blood volume. I believe that even small fistulae are accompanied by a minor dilatation of the heart and vessels and by commensurate increases in total blood volume, but our methods are not exact enough, and our eyes not keen enough, to demonstrate, and to recognize, such minor dilatations and such small increases in blood volume. It is freely granted that

the dye method is not an absolutely accurate method of studying blood volume, but one cannot ignore these results obtained in animals of the same litter on successive occasions, nor can one escape the fact that if there is a considerable increase in the capacity of the circulatory system by dilatation, there must be an increase in blood volume to fill it.



GRAPH 2.—Kymographic record of carotid blood pressure in the presence of bilateral femoral fistulae: (A) Both fistulae open; pulse 102. (B–C) Right fistula closed; pulse 87. (D–E) Both fistulae open; pulse 98. (F–G) Left fistula closed; pulse 82. (H–I) Both fistulae open. (J–K) Both fistulae closed simultaneously; pulse 76. (L–M) Both fistulae opened simultaneously. The effect upon blood pressure and pulse depends upon amount of blood diverted into the venous bed.

The Effects of Bilateral Femoral Fistulae on Arterial Pressures.—In an animal in which bilateral femoral fistulae had been established, October 31, 1934, the following observations (Graph 2) were made, June 28, 1938: Under nembutal anesthesia, and with aseptic precautions, the right carotid artery was cannulized and both fistulae in the groin were exposed to permit complete closure of the artery proximal to the fistula. On closing the right fistula, the blood pressure rose immediately from 124 Mm.Hg. (pulse 102) to 146 Mm. Hg., dropping to a level of 136 Mm.Hg. (pulse 87) as long as the fistula remained closed. On opening the fistula, the blood pressure dropped precipitately to 118 Mm.Hg., rising promptly to a level of 122 Mm.Hg. as long as

the fistula remained open. Similar figures were obtained on closing the left fistula (Graph 2). On closing both fistulae simultaneously, the pressure rose precipitately to a high point of 156 Mm.Hg. (pulse 76), dropping to a level of 140 Mm.Hg. as long as the fistula remained closed. On opening the two fistulae simultaneously, the pressure dropped to a low point of 100 Mm.Hg. for several beats only, rising promptly to the previous level of 124 Mm.Hg.

This was a convincing demonstration of the fact that the physiologic effects of opening and closing a peripheral fistula depend upon the volume of blood escaping into the shorter circuit.

The Effects of Bilateral Femoral Fistulae on Vena-Caval Pressures.—On April 14, 1939, the animal in which bilateral femoral fistulae had been established three and one-half years previously was again subjected to nembutal anesthesia and under aseptic precautions the following procedures were carried out: Through an incision in the flank the right kidney was mobilized, the renal artery was ligated, and a glass cannula, connected with an upright glass tubing of equal caliber, was inserted into the vena cava through the renal vein. The pressure, oscillating with respiration, was recorded in centimeters of water under the following conditions:

	Vena-Caval Pressure in cc. of Water Varying with Respiration		
Both fistulae open	12 –13	17 –18	19 –20
Right fistula closed			16.5–17
Left fistula closed	10 –11	15 –16	
Both fistulae closed	8.5– 9	12 –13	15 –15.5
Both fistulae open	12.5–13	15.5–16.5	18.5–19

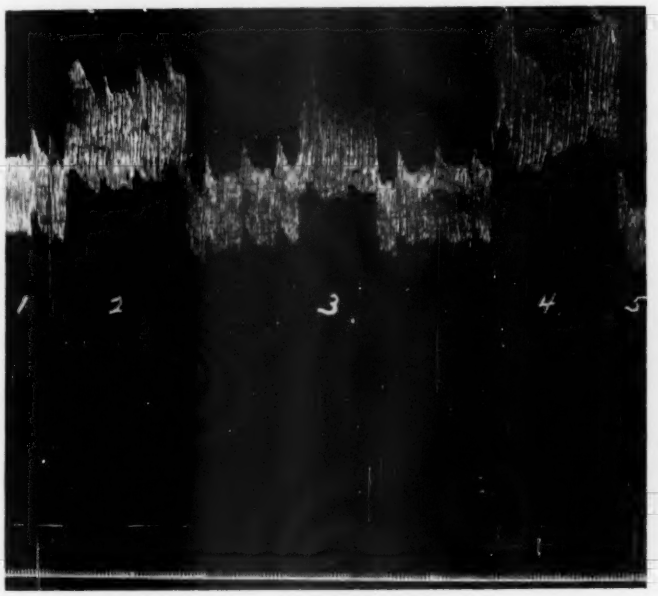
It was apparent, from observation, that the vena cava was able to accommodate much of the increased volume of blood transmitted through the fistula under arterial pressure by an increased velocity of flow. However, it was also apparent that the vena-caval pressure varied directly with the volume of blood diverted through the fistulae, being greatest with both fistulae open, least with both fistulae closed, and intermediate rises in vena-caval pressure occurred as one or the other fistula was opened separately.

On March 19, 1940, a second dog (Animal 3), in which bilateral femoral fistulae had been established on June 30, 1938, was investigated as to vena-caval pressures. Under morphine and ether, a long glass cannula was introduced through a rent in the left external jugular vein down into the thoracic vena cava. This experiment differed from the preceding experiment in that both the abdomen and the thorax were intact. The following observations were noted, the vena-caval pressures oscillating 5 to 10 Mm. with respiration:

	Vena-Caval Pressure in cc. of Water	Pulse
Both fistulae open	9.5–10	108
Right fistula closed	6.5– 7	90
Left fistula closed	7.5– 8	96
Both fistulae closed	5.5– 6	88
Left fistula reopened	7.5– 8	
Both fistulae open	9.5–10.5	

Again it was demonstrated that vena-caval pressure was increased in the presence of bilateral femoral fistulae, the greatest increase occurring when both fistulae were open. Closing one or the other fistula lowered the venous pressure 2–3 cc., whereas closure of both fistulae simultaneously lowered it 4 cc.

On March 4, 1940, the dog in which two femoral fistulae had been established on October 31, 1934, was again anesthetized, the remaining left renal vein was cannulized for vena-caval pressures, and the remaining left carotid



Conditions at Site of Fistulae	Mean Arterial Pressures Carotid Artery mm. Hg.	Vena Caval Pressures cm H_2O
1. Both Open	136	13 – 15
2. Left Closed	160	9 –10.5
3. Right Closed	156	8.5–10
4. Both Closed	182	6 – 7
5. Both Open	120	13 – 14

GRAPH 3.—Kymographic record of carotid arterial pressures dependent upon conditions at site of bilateral femoral fistulae of five and one-half years' duration. Vena-caval pressures obtained by cannulization of renal vein.

artery was cannulized for arterial pressures (Graph 3). It is evident from the kymographic record that when both fistulae were open the vena-caval pressure was high, and carotid pressure was low; when both fistulae were closed, vena-caval pressure was low and carotid pressure was high. Intermediate arterial and vena-caval pressures were obtained depending on closure of one or the other fistula.

From these many observations one may conclude: (1) That a rise in venous pressure occurs proximal to an arteriovenous fistula; and (2) that the extent of this rise in venous pressure depends upon the amount of blood diverted into the shorter circuit, and, therefore, upon the size of the fistula.

SUMMARY OF EXPERIMENTAL OBSERVATIONS

In the first 24 to 48 hours after the establishment of a large arteriovenous fistula, the heart diminishes in size, followed, if the animal survives, by a prompt return to normal, and, subsequently, by a gradual dilatation which may be apparent within four to five days.

Death, due to an excessive diversion of blood through the fistula, may occur, accompanied by a marked diminution in cardiac size.

The dilatation that accompanies an arteriovenous fistula is not restricted to the heart, but affects the vessels involved in the fistulous circuit. The same cause is responsible for both dilatations, an increase in the volume or bulk of blood flowing through that part of the circulatory system through which the blood short-circuited by the fistula must flow, namely, all the chambers of the heart, the proximal artery, fistula, and the proximal vein.

In the growing animal, the dilatation and enlargement may be very great without evidence of decompensation and may be accompanied by pronounced hypertrophy. It is suggested that when dilatation outstrips hypertrophy, decompensation occurs; when dilatation is paralleled by a commensurate hypertrophy, great enlargement and dilatation of the heart may occur without decompensation.

In a crucial experiment, involving three litter mates of equal weight and stature, one acting as control, one having an aorta-vena cava fistula 12 Mm. in circumference, and one having an aorta-vena cava fistula 18 Mm. in circumference, there occurred an increase in blood volume commensurate with the size of the fistula.

In the same animals, an increase in the capacity of the circulatory system occurred, also commensurate with the size of the fistula. The increase in capacity and the increase in blood volume closely paralleled each other.

In an animal with bilateral femoral fistulae the increase in blood pressure and reduction in pulse rate were greatest when both fistulae were closed simultaneously, and considerably less when either fistula was closed separately. The physiologic effect of a fistula, therefore, clearly depends upon the volume of blood diverted through the fistula and, therefore, upon its size.

The transient high systolic and diastolic pressures that persist for several days following operative closure of a fistula are due to the increase in blood volume that has occurred during the existence of the fistula. The permanent elevation of diastolic pressure is secondary to the elimination of an area of decreased peripheral resistance.

In animals having bilateral femoral fistulae, vena-caval pressures were highest with both fistulae open, least with both fistulae closed, and interme-

diate pressures were obtained on closing one or the other fistula separately. Venous pressures proximal to a fistula are determined by the volume of blood diverted through the fistula and, therefore, by the size of the fistula.

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DISCUSSION.—DR. JOHN HOMANS (Boston, Mass.): I shall address myself to the paper of Doctors Lee and Freeman. I have written out my discussion and I am sorry that I have done so, because the presentation with the pictures is so much more vivid and fascinating than I had anticipated from reading the paper alone.

The authors have called attention to the development of enlarged veins of a varicose type connecting with the malformed veins of the long cavernous hemangiomata of the limbs. I think this is not a very common combination, for I have seen four of these patients and only one of them suffered from this particular complication. That was a case which Dr. Arthur W. Allen, of Boston, very kindly allowed me to see, and that patient, who may, for all I know, have suffered discomfort and even fainting when her veins were allowed to fill as soon as she got out of bed, wore, at all times, a very tight stocking and kept her veins compressed. It would be very interesting to know whether, if she had failed to take care of herself, she would have developed this same sort of difficulty which Doctors Lee and Freeman describe. In most cases which I have seen, the enlarged varicose vein has been an integral part of the angioma, without evident superficial venous connections, and has required no particular treatment except for its unsightliness. If, however, one attempts to deal with one of these vessels he will discover that there is a very important arterial communication of some sort, it may be by very fine arterial twigs between the surrounding tissues, which are very much scarred, and the great vein itself. On one occasion, I attempted to

remove one of these veins and the patient not only bled on the table but bled seriously on the succeeding days. The wound failed to heal and the patient, during the next few months, bled a great deal more. Finally, it was possible, by going to the apex of the lesion, to find the vessel at the point where it pierced the deep fascia. If I had been able, in the first place, to inject a radiopaque substance and had secured a venogram, I should have made the whole procedure easier, but in my case it was possible to determine where this opening was, and by incising the deep fascia I finally discovered the plexus of large veins beneath it with which this large varicose vein communicated. I was then able to do away with back pressure from that source and afterwards the use of sclerosing solutions obliterated practically all of the serious varicosity.

So I agree with Doctors Lee, Freeman and Reid that the best way to attack these conditions is to find the connection with either the superficial varicose veins or with the deeper vessels, and by abolishing that connection subsequent treatment is made very much easier.

For some reason, which I do not understand, the lesions of the upper extremities are much less common than those of the lower. I think de Takats has published several cases recently. These cases apparently require treatment only because the individual is likely to injure the large vessel or to injure the angioma, but, of course, they are unsightly. I should think some of these could be excised.

Dr. James M. Mason (Birmingham, Ala.): World War No. 1 greatly stimulated interest in arteriovenous fistulae. As a result of the many wounds inflicted, there was a large amount of clinical material to be treated and studied. At this period, the fact was also established that cardiovascular disturbances resulted from the presence of these fistulae; and that these disturbances manifested themselves by local, peripheral, and central changes, some of which were not easy of explanation.

The immediate problem of the clinical surgeon was to give relief, if possible, by direct attack upon the local lesion, and to eliminate the fistulae. Various ingenious, and sometimes very difficult, operations have been devised for the accomplishment of this purpose.

It has been the problem of the cardiologist, the pathologist, and the experimental surgeon to investigate such phenomena as the blood pressure changes and bradycardiac phenomena observed on opening and closing a fistula; to explain the dilatation of the vessels proximal to the fistula, the hypertrophy and dilatation of the heart with decompensation and final congestive failure; and to account for the extensive collateral circulation which develops about the site of the fistula and makes quadruple ligation and excision of fistulae a safe operation in cases of long standing.

To the long list of contributors to the lucidation of these problems, Doctor Holman has added a brilliant piece of experimental work directed toward a better understanding of the most serious of these phenomena, namely, those that affect the blood volume, the arterial and vena-caval pressure and the degenerative changes in the heart.

In giving more careful study to his paper, and in comparing it with observations in certain of my clinical cases, I hope to arrive at an explanation of the question as to why patients with similar lesions react so differently in the matter of cardiac decompensation. Three of these have been reported (Mason, J. M.: Annals of Surgery, **107**, No. 6, 1029, June, 1938. *Idem: Ibid.*, **109**, No. 5, 735, May, 1939).

These fistulae resulted from gunshot and stab wounds in the subclavicular-axillary region: one on the right and two on the left side. The wounds

were approximately the same distance from the heart and were about the same diameter. One patient died from cardiac decompensation at the end of four days, without other injury or evidence of disease. Autopsy revealed a direct fistula with a wide lumen, between the subclavian vessels. The other two cases were under continuous observation of four and six months, respectively, before being operated upon. At no time did they show any evidence of cardiac embarrassment.

At operation which, in each instance, consisted of transvenous arteriorrhaphy, each case presented large varicosities in the region immediately adjacent to the fistula, indicating, to my mind, that obstruction to the free return of venous blood to the heart had taken place. In these two instances, hypertrophy may have kept pace with dilatation. The more likely explanation, however, is that the cause which resulted in production of the varicosities at the exact location of the fistula resulted in obstruction to the free return of blood to the heart, and thereby prevented its overfilling. In a previous communication, Holman has reported one case in which simple ligation of the vein proximal to the fistula resulted in obliteration of the thrill and caused the pulse to drop from 112 to 84. Harvey Stone reports a similar experience. Tixier and Arnulf have suggested that proximal venous ligation, some distance from the fistula, be employed in cases of great emergency, to relieve cardiac embarrassment until such a time as the patient may be able to undergo a curative operation.

Dr. Mont R. Reid (Cincinnati, Ohio): Since Doctors Lee and Freeman have referred to two of my cases, I thought it might be interesting to give you a little more of the story of one whom I followed for 17 years. This patient came to me as a young girl, age 13, with an extensive angioma of the left leg, extending up to the crest of the ileum, involving the perineum and the left labium. Her complaints were fainting on assuming the erect position and having frequent attacks of hemarthrosis of the left knee. Her symptoms, at first, were largely relieved by the use of an elastic stocking. Later, we began a long series of treatments which included ligations of veins and injections of sclerosing solutions. To-day, at the age of 30, she is so much improved that, recently, she married and is soon to have a baby.

I simply cite this case to stress the importance of the fact that something can be done to relieve these patients. It often requires a great deal of patience and persistence to accomplish it, but in this case it has been most gratifying, even though it has taken 17 years to get what now appears to be a satisfactory result.

In connection with Doctor Holman's paper, I must say that what he has shown here to-day appears to be most convincing. We have not been able to completely confirm his blood volume studies and I do not know why. We see a great many cases of arteriovenous aneurysms in which we cannot demonstrate a marked, or any increase in the total blood volume. I grant that we see very few, in which the heart is badly damaged, in which there is not an increase in the total blood volume. However, everyone knows that in cardiac decompensation, cardiac failure, or impending cardiac failure, there is an increase in the total blood volume. Whether it is entirely proven by Doctor Holman that the changes in the heart are due entirely to the handling of an increased blood volume, or whether or not the increase in the blood volume may be in some way connected with a decrease in function of the heart, we do not know.

We recently had a case of arteriovenous aneurysm in a young man with an aortic insufficiency. He had a very large heart and a markedly increased

blood volume. We are not sure which condition caused the increased blood volume; and this observation has started us studying cases of aortic insufficiency to see if they have an increased total blood volume.

I should like to ask Doctor Holman if he has undertaken any acute, or nonsurvival experiments and measured the cardiac output immediately after opening the fistula between the aorta and the vena cava. So far as our studies are concerned, there is an enormous increase in cardiac output immediately after producing such a fistula, and on measuring the amount of blood returned to the heart through the inferior vena cava, there is a tremendous increase in the amount of blood going immediately back into the heart.

Doctor Holman's illustrations showing a reduction in the size of the heart for a few days after the production of a fistula appear to leave no grounds for any doubt as to this effect. However, in our sacrifice experiments, with the chest open, we got the impression that there occurred, in a very few minutes, a definite increase in the size of the heart, and all studies showed a very marked increase in the cardiac output immediately after producing such a fistula.

Of course, Doctor Holman and I have argued this question many, many times. He is probably right. I have never been able to agree with him that simply because there is an increased vascular bed there must of necessity be an increase of blood volume.

Dr. Emile Holman (San Francisco, Calif., closing): I should like to ask Doctor Reid what fills this increased vascular bed—air? Humor? There must be something to fill this enlarged vascular bed and it is blood. Doctors Dock and Harrison, at the Peter Bent Brigham Hospital, in 1923, showed a doubling of cardiac output the moment we introduced a fistula into the circulatory system, and that was shown not only the day after the introduction of the fistula, but it was shown months later, with an increase in cardiac output during that time. There is no question about the increase in cardiac output the moment a fistula is introduced into the circulatory system.

Doctor Reid mentioned his acute experiments with the chest open. I do not believe they are comparable to experiments with the intact chest and the intact abdomen. Doctor Reid's experiments were also performed with a glass communication directly between the large vessels which did not permit bleeding into the distal venous bed, such as occurs in any arteriovenous fistula whether produced with a dagger, a bullet or by the experimentalist.

I also studied Doctor Reid's four clinical cases, in which he states that he did not get an increase in blood volume. A demonstrable increase in these cases would not be expected with present methods of determining blood volume. There was no increase in blood pressure or fall in pulse rate as I recall it, when he closed the fistula, and there was very little dilatation of the heart. This suggests that the fistulae in these four cases were of small size. With ordinary methods of determining blood volume we cannot demonstrate increases in small fistulae. I do believe, however, that any fistula of any size causes an increase in blood volume and a dilatation of the heart and vessels, but as our methods and instruments of precision are not sufficiently accurate, we cannot determine such small increases at the present time.

THE SURGICAL TREATMENT OF ANEURYSM OF THE ABDOMINAL AORTA*

REVIEW OF THE LITERATURE AND REPORT OF TWO CASES, ONE APPARENTLY SUCCESSFUL

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Clinically recognizable aneurysms of the abdominal aorta occur infrequently, but the fact that Kampmeier[1] was able to collect 65 proven cases of aortic, and three cases of celiac aneurysm from the records of the Charity Hospital in New Orleans, over a period of 30 years, indicates that they are not extremely rare.

The duration of abdominal aneurysms is difficult to determine, for they are deeply situated and are usually not suspected until they are far advanced. However, in 87.7 per cent of the 57 cases of Kampmeier's series, in which reliable data were available, symptoms had been noted for less than one year, and in 61.3 per cent of them, symptoms had been present for less than six months. Furthermore, death occurred within one month after admission to the hospital in 38 of the 68 cases. It is evident, therefore, that the disease is apt to run a rapidly fatal course.

Up to the present time, all forms of therapy have yielded poor results. Strictly conservative treatment offers little, and wiring, either with or without electrolysis, is at best only palliative. Judging from the literature, only a small number of surgeons have felt that direct surgical attack upon aneurysms of the abdominal aorta was justifiable, and it must be admitted that the results obtained by surgical intervention have been discouraging. However, a closer study of the reported cases reveals some cause for optimism. From 1817, when Sir Astley Cooper first ligated the abdominal aorta, to 1920, none of the 18 patients in whom the aorta (abdominal, 16, or lower thoracic, two) was occluded for abdominal aneurysm survived to leave the hospital. Since 1920, seven of the 12 patients in whom the aorta has been partially or completely obstructed for aneurysm have been discharged from the hospital as improved, and one other lived four and one-half months but did not leave the hospital (Table I). While it is not certain that any of these patients have been cured, the results demonstrate that occlusion of the aorta is not necessarily fatal. This information is important, for occlusion of the involved artery is necessary for the cure of most aneurysms. The only exceptions to this rule are found in those clearly sacculated aneurysms which may be treated by lateral ligature of the sac or by restorative aneurysmorrhaphy. Such

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TABLE I

LIGATIONS OF THE AORTA FOR ABDOMINAL ANEURYSM

PREVIOUS TO 1920

Surgeon	Year	Location of Aneurysm Sex and Age	Ligature Material	Level of Ligation	Survival Period	Cause of Death
1. Cooper[2]	1817	Left iliofemoral (male, 38 yrs.)	?	Distal portion of aorta	3 days	?
2. James[3]	1829	Left external iliac (male, 44 yrs.)	?	Distal portion of aorta	3½ hrs.	Shock
3. Murray[4]	1834	Right iliofemoral (male, 33 yrs.)	?	Distal portion of aorta	23 hrs.	?
4. Monteiro[5]	1842	Right iliofemoral (male, 30 yrs.)	?	Distal portion of aorta	10 days	Hemorrhage
5. South[6]	1856	Right common and external iliac (male, 28 yrs.)	?	Distal portion of aorta	43 hrs.	?
6. Stokes[7]	1868	Right iliofemoral (male, 50 yrs.)	Silver wire	Distal portion of aorta	12¾ hrs.	Shock
7. McGuire[8]	1868	Aorta and both common iliacs (male, 30 yrs.)	?	Distal portion of aorta	Few hrs.	?
8. Watson[9]	1869	Iliac (? ?)	?	Distal portion of aorta	65 hrs.	Gangrene
9. Milton[10]	1890	Aorta, ruptured (male, 45 yrs.)	?	Distal to renal arteries	24 hrs.	Shock and hemorrhage
10. Körte[11]	1899	Right external iliac, ruptured (male, 28 yrs.)	?	Distal aorta. (Sac opened 37 days after ligation of common iliac. Severe hemorrhage. Aorta ligated)	1 hr.	Shock and hemorrhage
11. Keen[12]	1899	Proximal abdominal aorta, ruptured (male, 52 yrs.)	?	Below diaphragm	48 days	Cutting through of ligature
12. Tillaux[13]	1900	Left iliac, ruptured (male, 56 yrs.)	?	Terminal aorta	39 hrs.	?
13. Morris[14]	1901	Midportion of abdominal aorta (female, 24 yrs.)	Soft rubber catheter and clamp forceps	Terminal aorta	?	Gangrene of bowel from pressure by forceps
14. Halsted[15]	1906	Extending from above inferior mesenteric artery to near diaphragm (male, 36 yrs.)	Aluminum band	Band applied to aorta below inferior mesenteric 23 days after one had been applied to thoracic aorta	18 days	Rupture of aneurysm

15. Halsted[16]	1908	Left common iliac (male, 58 yrs.)	Tape, after rupture of aorta while attempt was being made to apply aluminum band	Terminal aorta	12 hrs.	Hemorrhage and shock in patient with hypertrophied and dilated heart
16. Halsted[15]	1909	Aorta, extending from renal arteries to bifurcation (male, 53 yrs.)	Aluminum band	Between renal and superior mesenteric branches. (Partial occlusion)	47 days	Abscess developed at site of band. Death from results of infection
17. Halsted[17]	1911	Aorta, below renal arteries (elderly woman)	Aluminum band	Below renals	6 wks.	Hemorrhage from cutting through of band
18. Heuer[18]	1917	Abdominal aorta at level of celiac axis (male, 31 yrs.)	Aluminum band	Lower thoracic aorta. (Partial occlusion)	1 mo., 9 days	Rupture of aorta at site of band
			SUBSEQUENT TO 1920			
19.* Vaughan[19]	1920	About origin of superior mesenteric artery (male, 39 yrs.)	Cotton tape (½ inch)	Distal to aneurysm below inferior mesenteric artery. (Partial occlusion)	2 yrs., 1 mo.	? (Aneurysm much larger but had not ruptured)
20. Reid[20]	1921	About level of inferior mesenteric artery (male, 36 yrs.)	Cotton tape	Proximal to celiac artery. (Previously partly occluded between inferior mesenteric and renal arteries)	4½ mos. (after first operation)	Rupture of aneurysm
21. Watts[21]	1923 (Feb. 26)	From superior mesenteric artery to near bifurcation (female, 28 yrs.)	Cotton tape	Just above superior mesenteric artery	22 hrs.	Renal arteries came off of sac. Patient developed suppression of urine. Ligature was loosened about 3 hrs. before death but pulsation did not return to sac
22.* Matas[22]	1923 (Apr. 9)	Terminal aorta (female, 28 yrs.)	Cotton tape	Below inferior mesenteric artery	1 yr., 5 mos.	Massive, tuberculous, pulmonary hemorrhage. (Aneurysmal sac almost obliterated)
23.* Watts[21]	1923 (Sept. 15)	Below superior mesenteric, extending almost to bifurcation (female, 60 yrs.)	Cotton tape	Below superior mesenteric artery. (Partial occlusion)	3½ yrs.	Rupture of aneurysm (?). No autopsy
24.* Brooks[23]	1925	Terminal aorta (male, 59 yrs.)	Fascia lata (proximal) and heavy silk (distal to fascia)	Distal to inferior mesenteric artery	About 3 mos.	Intestinal obstruction. (Aneurysmal sac obliterated)

TABLE I (*Continued*)

Surgeon	Year	Location of Aneurysm Sex and Age	Ligature Material	Level of Ligation	Survival Period	Cause of Death
25. Reid[24]	1928	Aorta at level of celiac axis (female, 35 yrs.)	Insertion of fascial plug with fixation by silk sutures	Lower thoracic aorta	12 hrs.	Hemorrhage from divided intercostal artery
26. Andrus[25]	1929	Lower epigastric region (male, 57 yrs.)	Cotton tape	Artery doubly ligated and divided between tapes in lower thoracic region	1½ hrs.	Shock
27.* LaRoque[26]	1929	Right common iliac (female, 30 yrs.)	Umbilical tape and heavy silk	Distal abdominal aorta. (Subtotal occlusion)	?	Alive and greatly improved at end of 14 mos.
28. Bigger[27]	1938	From inferior mesenteric to bifurcation of aorta (male, 54 yrs.)	Fascia lata	Proximal to inferior mesenteric artery	18 hrs.	Left-sided heart failure. Pulmonary edema
29.* Bigger[27]	1938	Level of inferior mesenteric (male, 25 yrs.)	Fascia lata	Proximal to inferior mesenteric artery. Endo-aneurysmorrhaphy 1 mo. later		Alive and apparently well
30.* Elkin[28]	1939	Terminal aorta (male, 50 (?) yrs.)	Cotton tape	Distal to inferior mesenteric artery. (Subtotal occlusion)		Alive and at work, 11 mos. later

* Successful case.

aneurysms are not common in any location and are particularly rare in connection with the abdominal aorta.

If the foregoing statements are true, the following problems need to be clarified: (1) Under what circumstances may the aorta be occluded with reasonable chance of success; (2) what method of occlusion is preferable; and (3) is proximal occlusion curative? If not, is the Matas operation (obliterative endo-aneurysmorrhaphy) feasible in spontaneous aortic aneurysm? None of these questions can, as yet, be answered with any assurance but some important evidence bearing on them should be given consideration.

In regard to the circumstances under which aortic occlusion may be considered justifiable, it seems obvious that in elderly or greatly debiliated patients the chances of success are diminished and it is doubtful if such an operation should be undertaken on very poor-risk patients. If operation is undertaken, it is almost certain that the occlusion should be accomplished in more than one stage. Extensive, diffuse calcification of the aorta should probably be considered a contraindication to surgery.

The portion of the aorta involved and the importance of the branches arising from the aneurysmal sac have an important bearing on the prognosis. For example—it is reasonable to suppose that aneurysms arising from the aorta distal to the renal arteries will be more susceptible to surgical cure than those arising proximal to these vessels. This would appear likely because the effect on the circulatory apparatus is less pronounced the farther the lesion is from the heart, and, more important still, because there is danger of occlusion of essential arteries such as the superior mesenteric, the celiac, or the renals when the proximal portion of the abdominal aorta is involved. It has been shown by Halsted and Reid that the aorta may be occluded above the renal arteries, but if that portion of the aortic wall from which the superior mesenteric, the celiac, or the renal arteries arise is involved in the aneurysmal sac, ligation of the aorta will, almost certainly, result in occlusion of the origin of these essential vessels by clot and, thus, will result in an insufficient circulation to the organs supplied by them. Stated in a different way, the collateral circulation may be sufficient to nourish the stomach, intestines, and kidneys when the aorta is obstructed proximal to the origin of the celiac, superior mesenteric, or renal arteries, but only through the lumen of the aorta distal to the obstruction. It is, therefore, probably wiser to discontinue an operation when any of these vessels is found to arise from the wall of the aneurysmal sac. This apparently does not apply to the inferior mesenteric artery, for, as demonstrated by Archibald,[29] the main trunk of this artery may be occluded without producing necrosis of that portion of the intestine supplied by it.

A study of the results of occlusion of the aorta by the various materials now in use convinces one that none of them is satisfactory and there is no unanimity of opinion as to which one is best. Most of the reported failures may be attributed to one or the other of the following causes: (1) Either the ligatures rapidly cut through the wall of the aorta and cause fatal hemorrhage;

or (2) they fail to produce a permanent occlusion of that vessel. Fascia has many advantages for temporary or preliminary partial occlusion, but the results are not permanent. Matas recommends cotton tape, and it is probably significant that it was the material used in four of the seven more or less successful cases so far reported, but even wide tape may cut through the aortic wall and probably always gives, to some extent, after a few days.

In all except a few cases of aneurysm of the abdominal aorta which have been operated upon, the surgical procedure has been limited to an attempt to occlude the aorta, usually proximal to the origin of the sac. Proximal occlusion of the aorta is undoubtedly an essential part of the operative procedure in most cases, either preliminary to, or as a part of some more extensive operation, but there is considerable reason to doubt that proximal ligation alone will cure an appreciable number of cases even if the occlusion can be made complete and permanent. It is known that occasionally an aneurysmal sac spontaneously becomes filled with clot and is obliterated. This naturally occurs in a certain percentage of cases following proximal occlusion, and Halsted,[15] Matas,[30] Reid[31] and Holman[32] have obtained such results in iliac and femoral aneurysms, but it does not seem reasonable to suppose that it will happen frequently. Brooks[23] obtained complete obliteration of an aneurysm of the terminal aorta following proximal occlusion of that vessel but points out that this result probably occurred because the common iliac arteries were largely obstructed by pressure from the aneurysmal sac. The condition was, therefore, essentially similar to that produced by proximal and distal ligation. If there is a sufficient collateral blood flow to nourish the tissue distal to the obstruction, there is apt to be sufficient reflux flow to prevent complete and permanent obliteration of the aneurysmal sac. It would seem, therefore, that proximal occlusion of the aorta should be undertaken as a preliminary to more extensive procedures in suitable cases. In cases not suitable for other procedures, proximal occlusion may be valuable for palliation.

In carefully selected cases of abdominal aortic aneurysm, it would seem wise to perform a preliminary partial or subtotal proximal occlusion of the aorta; then, after a suitable delay, perform aneurysmorrhaphy or possibly aneurysmectomy. Unfortunately, it is probable that few cases are suitable for either of these procedures. Thus far, they have been attempted in only a small number of cases, some of which were obviously not suitable.

Bogoraz[33] reported a case which he described as an arteriovenous aneurysm between the abdominal aorta and the left renal vein. The aneurysmal sac, which was about the size of a goose egg, arose at the site of origin of the left renal artery from the aorta. A proximal ligature was applied between the sac and the aorta and a distal ligature was then applied to the renal artery, and the sac excised. The kidney was also removed. The patient recovered. Although this is described as an aortic aneurysm, the fact that he was able to ligate the neck of the sac makes it appear likely that it originated from the

base of the left renal artery which may have been greatly dilated as a result of the fistula.

Brooks[34] attempted excision of a large thin-walled aneurysm of the terminal aorta but this had to be abandoned because of rupture of the sac followed by excessive hemorrhage. The aorta was then ligated proximal to the aneurysm and, as was previously pointed out, this resulted in complete obliteration of the sac. So far as can be determined, there has not been a successful excision of a true aortic aneurysm.

Aneurysmorrhaphy for the cure of aneurysm of the abdominal aorta has been attempted by Lozano,[35] Munro,[36] Crile,[37] Kümmel[38] and Bigger (traumatic). All of the patients so operated upon, except the one reported in this paper, died. A report of Crile's case has not been available for study, but a review of Munro's, Kümmel's, and Lozano's reports brings out certain points in the technic employed in each of these operations which may be significant in relation to the outcome.

Munro did not provide for occlusion of the aorta either proximal or distal to the aneurysm, so when the thin sac was torn while it was being freed up he had to control hemorrhage by applying a stomach clamp tangentially. He then attempted to obliterate the sac by suture while the clamp was in place. This was naturally less satisfactory than a more deliberately executed intrasaccular suture of the vascular openings. Munro's patient died from hemorrhage the night following operation.

Kümmel approached an aneurysm of the proximal portion of the abdominal aorta through the chest wall and diaphragm. The aorta was compressed proximally but not distally when the sac was split open. Apparently, the intrasaccular suture was reasonably satisfactory but after the repair had been completed a gauze tampon was inserted between the aorta and spine. This caused bleeding which was difficult to control and Kümmel blamed his failure on this maneuver. However, it is probable that there was considerable unnecessary hemorrhage from the distal aorta during the primary suture and that this played an important part in the unfortunate outcome in this case.

Lozano applied clamps (without rubber coverings) to the very sclerotic aorta above and below the aneurysm before opening the sac. He then closed the arterial openings in the sac by suture and obliterated it. This part of the operation was apparently successfully accomplished, but when the obstructing clamps were removed there was bleeding where the sclerotic vessel wall had given way beneath them. It is possible that, with such advanced sclerosis, any form of compression would have produced the same result, but unprotected clamps would be particularly likely to cut through such a vessel wall.

Careful provision for the control of hemorrhage is essential in either excision or intrasaccular suture of aneurysm. In peripheral aneurysms this is best accomplished by the use of a tourniquet but when large arteries of the trunk are involved the main vessel must be occluded both proximally and distally and, in addition to this, collateral vessels entering the sac should be

ligated when possible before the sac is opened. If these precautions are not taken, excessive hemorrhage is apt to occur.

There are many unsolved problems in connection with the surgical treatment of aneurysms of the abdominal aorta. Some of them may be solved by carefully controlled animal experiments, but others can be solved only by observations on patients with aortic aneurysms. For this reason it is essential that such patients, especially those subjected to operation, be carefully observed and that the results be reported in detail.

With this in mind, the author presents the following two cases:

CASE REPORTS

Case 1.—R. E. C., white, male, age 54, was admitted to the Memorial Hospital, Richmond, Va., February 11, 1938, complaining of a pulsating mass in the abdomen and pain in the right hip. He had noticed the abdominal pulsation about eight months before but the pain in the right hip was of only three weeks' duration. His past history was not significant.

Physical Examination.—The patient was a frail, moderately emaciated white male, who appeared to be about 60 years of age. He was inclined to remain in the dorsal recumbent position with the right thigh flexed on the abdomen at an angle of approximately 90°. The abdomen and lower extremities presented the only significant findings. Abdominal examination revealed a firm, pulsating mass extending across it at about the level of the umbilicus, more prominent to the left of the midline. There was also a sausage-shaped mass in the right lower quadrant which was quite tender. There was atrophy of the muscles of both legs. No edema.

His temperature fluctuated between 99° and 101° F. Blood pressure 100/50. R.B.C. 2,880,000, Hb. 60 per cent; W.B.C. 12,500, 78 per cent polymorphonuclear neutrophils. Wassermann negative. Blood sugar 91, nonprotein nitrogen 34. Urine negative. *Clinical Diagnosis:* Aneurysm of the abdominal aorta.

The moderate elevation of temperature, leukocytosis, and increasing pain were erroneously interpreted as being due to a retroperitoneal rupture of the aneurysm, so it seemed wise to obstruct the aorta proximal to the aneurysm.

Operation.—February 21, 1938: Under general anesthesia, the right thigh could be easily extended, indicating that its flexion had been due to spasm of the iliopsoas muscle. The abdomen was entered through a right paramedian incision extending from the mid-epigastrium to the pubis. It was found that the aneurysm extended considerably above the most prominent, externally palpable portion of the sac, arising about 2 cm. below the third portion of the duodenum at the origin of the inferior mesenteric artery. The second aneurysm involved the right iliac artery, and a poorly defined mass extended laterally and upward beneath the terminal ileum and cecum. The posterior peritoneum was carefully incised just proximal to the aneurysm. The root of the mesentery was separated up for a short distance and retracted to the right, thus exposing the third portion of the duodenum which was retracted upward, to expose the aorta. An aneurysm needle was passed around the aorta just above the aneurysm and a strand of chromic catgut drawn around the vessel. A segment of fascia lata, about five inches in length and about 1 cm. in width, was removed from the right thigh, attached to the catgut ligature, and pulled around the aorta. The strip of fascia was then tied down sufficiently tightly to cause complete disappearance of all pulsations distal to it and the knot fixed by a heavy silk suture-ligature. A doubled, heavy silk ligature was left around the aorta for future identification. It was not tied. The vena cava was not occluded.

Blood pressure was 95/60 when the operation was started and remained remarkably constant throughout the procedure, showing no change when the aorta was occluded. When he was returned to the ward from the operating room his blood pressure was

90/50 and it remained at approximately this level for ten hours. It then began to fluctuate and the systolic pressure ranged between 120 and 60.

Postoperative Course.—Immediately following operation, both feet and legs were warm but the right foot was somewhat paler than the left. About six hours after operation, the right foot and leg became pale and cold, but the color and temperature of the left one remained good, and a faint pulse was palpable in the left femoral artery. The right femoral artery remained pulseless; and, at the end of 12 hours, the right leg was becoming shriveled in appearance and showed areas of discoloration over the dependent portions. It seemed obvious, at this time, that the circulation in the right leg was inadequate, therefore, the right femoral vein was ligated, with the hope that this might improve the circulatory balance in that leg.

Twelve hours after the first operation, his systolic blood pressure fell to 60, and 500 cc. of whole blood was administered. His pressure rose to 104 but two hours later dropped to 90, and remained at that level until an hour before his death. He developed edema of the lungs and died 18 hours after ligation of the aorta.

Autopsy.—An aneurysm of the distal portion of the abdominal aorta and another of the right common iliac artery were found, both apparently due to arteriosclerosis. The ill-defined mass in the right side of the abdomen was a large retroperitoneal abscess. He also had generalized arteriosclerosis, chronic interstitial nephritis, occlusion of the right ureter with pyelonephritis and associated venous thrombosis, a large soft spleen, and other signs of severe sepsis. The immediate cause of death was left-sided heart failure with pulmonary edema. When the aorta was opened the site of occlusion was found to be sufficiently patent to permit the passage of an ordinary probe, but the aneurysmal sac was largely filled by fresh clot. The distal portion of the iliac aneurysm was filled by old clot and its proximal portion by fresh clot. The lumen of the external iliac artery was occluded by advanced arteriosclerotic changes and a fresh thrombus filled the internal iliac (hypogastric) artery.

Comment.—Since the autopsy showed long-standing occlusion of the external iliac artery, the acute circulatory deficiency following aortic occlusion is adequately explained by the formation of a clot in the hypogastric artery and its major branches, for, under the circumstances, this vessel was an essential collateral channel. Failure of the left side of the heart probably resulted from the additional burden imposed upon a cardiac musculature greatly weakened by prolonged sepsis. Occlusion of the aorta, *per se*, probably played an unimportant part in the outcome, and abdominal exploration without aortic occlusion might well have led to the same result.

Case 2.—C. C., white, male, age 25, was admitted to the Memorial Hospital, December 13, 1938, at 3:30 o'clock P.M., and stated that he had felt well the previous day but at about 6:00 o'clock A.M., on the day of his admission, he leaned over to wash his face, became dizzy, and fainted. After he regained consciousness he had severe abdominal pain, most marked in the midepigastrium and radiating to the region of the left shoulder blade. He vomited once and had a bowel movement, but the severe pain persisted. When first examined by a physician he was pulseless and complained of extreme thirst.

Physical Examination.—Temperature 101° F., pulse 140, respirations 22, blood pressure 50/30. He was very pale. There was no cyanosis and no distention of the neck veins. Heart rate was rapid, rhythm regular, no murmurs. His pupils were constricted. The abdomen was rigid and apparently contained fluid. *Tentative Diagnosis:* Ruptured abdominal aortic aneurysm. This was considered especially likely because of the following history:

On September 7, 1937, this patient was admitted to the Galax Hospital, Galax, Va., for the treatment of multiple buckshot wounds. One shot had entered below the crest of

the right ilium and had coursed completely across the body and was lying in the left side near the anterior end of the eleventh rib. The patient was badly shocked and presented the signs of intra-abdominal injury. At operation no intestinal perforation was found but there was a massive retroperitoneal hemorrhage, the origin of which was not determined. He was discharged from the hospital 14 days later, and had apparently been in good general health until the onset of the present trouble.

A short time after admission to the Memorial Hospital he was given 450 cc. of blood by the multiple syringe method, following which his condition showed marked improvement. At 8:00 o'clock P.M., four and one-half hours after his admission, his blood pressure was 130/82, pulse 112, temperature 101.6° F. Although his abdomen was rigid and distended, it was thought that a pulsating mass could be felt about one inch to the left of the umbilicus. Pulsation in the vessels of the lower extremities was full and equal.

Before transfusion his blood showed R.B.C. 3,280,000, Hb. 58 per cent; W.B.C. 30,000, 92 per cent polymorphonuclear neutrophils, Wassermann and Kline reactions negative. The following morning his blood findings were R.B.C. 2,900,000, Hb. 44 per cent; W.B.C. 21,500, 92 per cent polymorphonuclear neutrophils. His urine was negative.

At 8:30 o'clock on the morning after admission his blood pressure was still normal and his pulse of good volume. At 8:45 o'clock, 15 minutes later, he had another attack of syncope and his pulse and blood pressure could not be obtained. The foot of his bed was elevated and he was given 600 cc. of blood by the multiple syringe method. After this attack of syncope his abdomen became greatly distended and there was definite evidence of free fluid in the peritoneal cavity. The patient complained of very severe, continuous abdominal pain. Exploration of the abdomen in an attempt to control the bleeding was decided upon.

Operation.—At 4:00 o'clock P.M. the patient was given 150 mg. of novocain intraspinally. At the time of the intraspinal injection his blood pressure was 110/70, but it dropped rapidly and when the abdomen was opened his systolic pressure was only 70 Mm.Hg. Shortly thereafter it was impossible to determine it. The old left rectus scar, the result of his operation 15 months previously, was excised and the incision lengthened both above and below so that when completed, it extended from about two inches below the left costal margin to about two inches above the pubis. A massive intra-abdominal hemorrhage, partly clot and partly liquid blood, was found. About 500 cc. of this blood was removed, citrated, filtered, and administered intravenously. In addition to this, he was given 500 cc. of blood from a donor, the entire liter of blood being administered during the course of the operation. The bleeding had come from the rupture of an aneurysm of the lower portion of the abdominal aorta but there was no bleeding when the abdomen was opened, as the tear in the anterior wall of the aneurysm was plugged by a firm clot. Although the bleeding had temporarily stopped, it was felt that the aorta must be obstructed proximal to the aneurysm to prevent a recurrence of the hemorrhage. The aneurysm, which was about 8 to 10 cm. in diameter, arose from the anterior wall of the abdominal aorta at the level of the inferior mesenteric artery.

An incision was made through the posterior peritoneum immediately above the aneurysm, and the third portion of the duodenum was exposed, freed up, and retracted upward to expose the aorta (Fig. 1). The aorta was separated from the vena cava, and an aneurysm needle carrying a heavy catgut ligature was carefully passed around the artery; three heavy catgut ligatures were then tied to the first one and drawn around the vessel. A strip of fascia lata about three-quarters of an inch wide and six to eight inches long was removed from the left thigh, attached to one of the catgut ligatures, and drawn around the aorta twice. It was tightened until all distal pulsation ceased, and fixed by silk sutures. A long piece of heavy silk was left in place around the aorta for future use. Before the aorta was occluded a pronounced, continuous thrill could be felt in one of the adjacent vessels. This thrill could not be felt after the aorta was occluded, so its site was never definitely determined; but it was thought that it was an arteriovenous fistula, probably involving the mesenteric vessels.

Immediately following occlusion of the aorta the systolic blood pressure rose sharply to 180 Mm.Hg., and the patient had a mild convulsive seizure. Two hours after his return to the ward he was able to move his arms and legs. His feet and lower legs were cold, pale, and the veins were collapsed, but the upper legs and thighs were warm and of good color.

Postoperative Course.—The following morning (December 15) his general condition was satisfactory, systolic blood pressure 140, lower extremities were warm, and the color

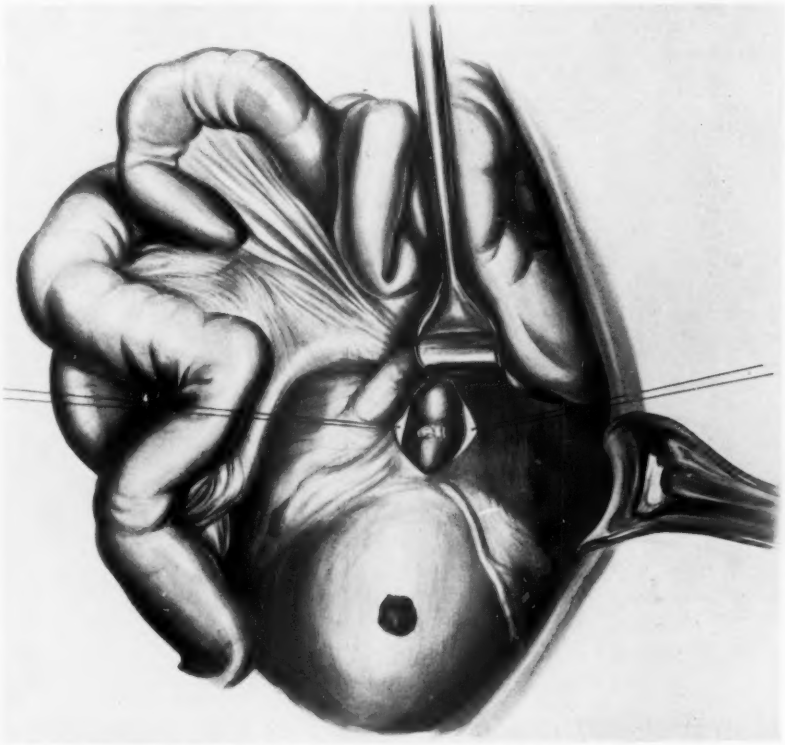

FIG. 1.—Case 2: Drawing showing point of rupture of aneurysm and level of occlusion of the aorta.

of his feet was much improved, but no pulsations could be made out in any of the arteries of the lower extremities, including the femorals. It was discovered, however, that during the night the patient had developed complete paralysis of both lower extremities. There was also marked diminution in tactile sensation and widespread involuntary muscular twitching. These findings suggested involvement of the nerves as a result of marked temporary anemia.

Saline absorption by Dr. Nathan Bloom on December 15, the day after operation, was as follows:

	Right Leg	Left Leg
Inner side of foot	20 minutes	15 minutes
Inner side of midportion of lower leg	30 minutes	25 minutes
Patella	30 minutes	25 minutes
Midthigh	30 minutes	—

On December 16, he complained of numbness and tingling of his lower extremities but was still unable to move them.

On December 17, sensation had not returned to the lower legs but he was able to flex his knees slightly.

On December 20, he began to complain of burning and aching in his legs, and for several days this was so severe as to require opiates for its relief. He was unable to void until the fourth postoperative day.

Neurologic examination by Dr. Gayle Crutchfield, December 22, showed that all of the deep reflexes of the lower extremities were abolished except the right prepatellar, which was hypo-active. Plantar response was normal. All muscle groups showed greatly diminished power. In addition to the above findings there was hypesthesia below both knees, extending down to and including a part of the dorsum of each foot. His impression was that the neurologic changes were the result of temporary anemia of the peripheral nerves of the lower extremities.

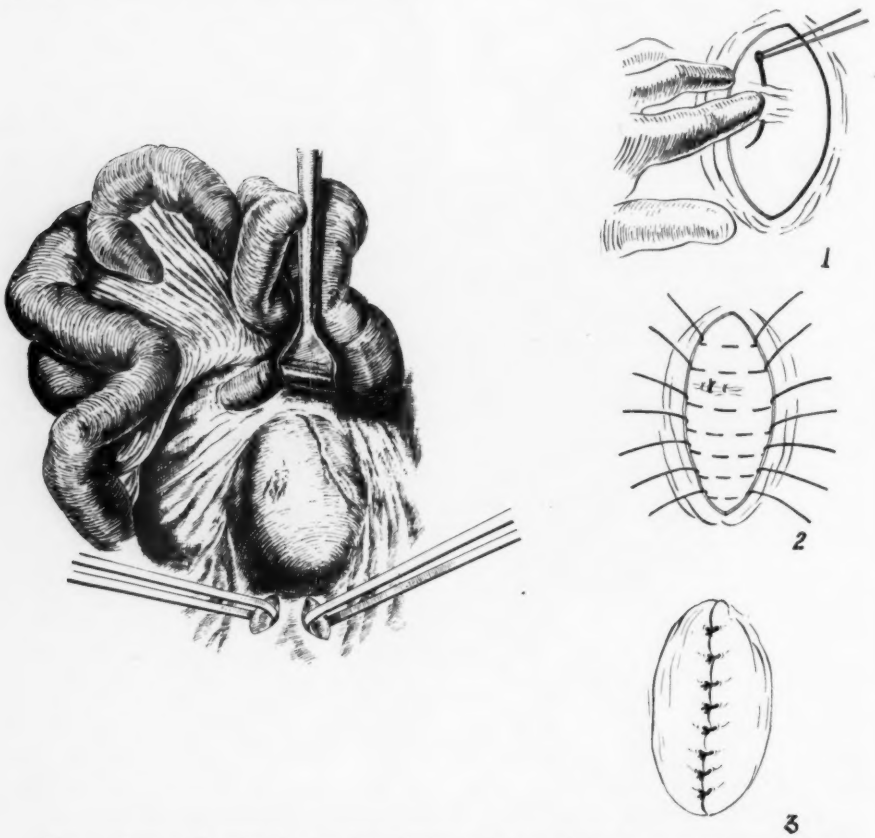

FIG. 2.—Case 2: Drawing showing the reduction in the size of the aneurysm one month after the occlusion of the aorta. (1) The method of occluding the opening between the aorta and aneurysmal sac. (2 and 3) Obliteration of the aneurysmal sac.

On December 23, the patient was able to move his feet and toes, and was conscious of the sense of pressure, but the finer senses of touch and slight pain had not returned. Muscular twitching had completely disappeared.

On January 4, 1939, pulsation was detected on both sides just above the inguinal ligaments. This was thought to be in the deep epigastric arteries, as no pulsation could be made out below the inguinal ligaments. Sensation and motion were both greatly improved, but he still complained of pain in the thighs, knees, and ankles.

Second Operation.—On January 10, the patient was again operated upon. Under general anesthesia, the left rectus scar was again excised with especial care to avoid injury to the left deep epigastric artery, an important collateral vessel. The omentum

was adherent to the parietal peritoneum but the bowel was free. The aneurysmal sac was reduced to about one-half its previous size and was not pulsating. The iliac arteries were not pulsating. A loop of small bowel adherent to the area at which the sac had ruptured was freed up without particular difficulty. The heavy silk ligature which had been placed around the aorta proximal to the aneurysm was found and preserved so that it could be used to control bleeding from above. Both common iliac arteries were exposed and heavy silk ligatures placed around them for the control of retrograde hemorrhage (Fig. 2). After these provisions had been made for the control of bleeding, the aneurysmal sac was opened in a perpendicular direction and several pieces of tissue were removed from its wall for microscopic study.

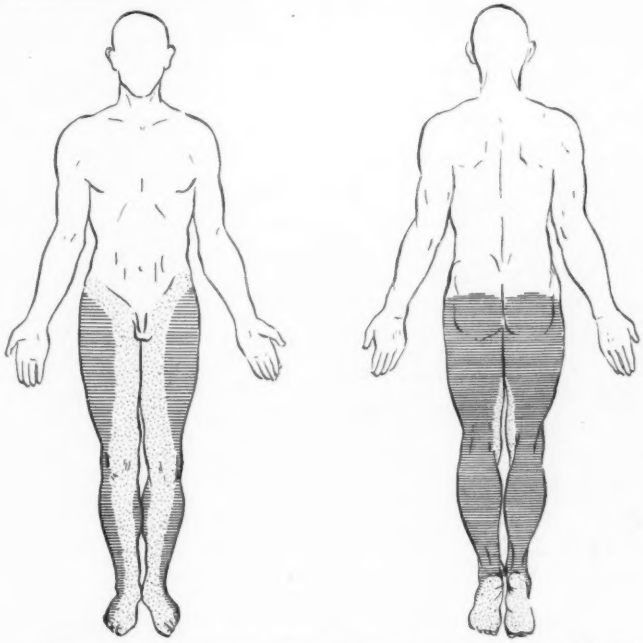

FIG. 3.—Case 2: Sensory changes following occlusion of the abdominal aorta. The dotted areas indicate slight hypesthesia to pain and temperature. The transverse lines indicate the areas of marked hypesthesia to pain and temperature.

Pathologic Examination.—Dr. George Z. Williams: "The specimen submitted for examination consists of several small pieces of dense, pink tissue measuring about 2.5 cm. in greatest length and obviously from the wall of some type of sac.

"*Microscopic Examination.*—The paraffin imbedded and hematoxylin-eosin stained sections taken from this material reveal the characteristic structures of arterial wall. However, there is marked degenerative change with much necrobiosis in which the fibers of elastic tissue can still be identified but distortion caused by sclerosis and collagenous increase is marked. There is no sharp demarcation between the degenerating sclerotic arterial walls and the superimposed hyalinized platelet thrombus which also shows degenerative changes. Infiltration of chronic inflammatory cells, particularly monocytes, around small patches of hemorrhage is seen in various portions of the arterial wall. Some evidence of old hemorrhage is seen in the presence of cells containing hemosiderin pigment and these are scattered through the fibroblastic sclerotic areas. No specific inflammatory changes can be seen. These changes are typical of a severe sclerotic lesion of the arterial wall with old and recent hemorrhages in the wall which are undergoing healing changes."

A considerable quantity of blood clot was removed from the sac without encounter-

ing hemorrhage, but when a dense piece of clot was removed from the upper right posterior wall of the sac, overlying the anterior portion of the aorta, bright red blood came out under considerable pressure. It was possible to control this by placing the distal phalanx of the left index finger in the communicating opening. A silk suture was passed deeply through the lower edge of the opening beneath the finger, then out through the upper edge. Large bites of tissue were included on both sides of the opening and when tension was made on this suture it was found that bleeding was largely controlled. When additional sutures were placed on each side of it there was no further bleeding even when the silk loops around the aorta and the common iliac arteries were released. The aneurysmal sac was obliterated by six silk sutures passed from the left, through the posterior wall of the sac and then through the upper right side of the sac. The margins of the sac were approximated with interrupted sutures of silk and the omentum was tacked over the suture line.

At this second operation it was found that the lower sigmoid and rectum were firmly adherent to the walls of the pelvis, possibly due to circulatory changes in the bowel following occlusion of the aorta.

At the completion of the operation the patient's general condition was good. Systolic blood pressure 130; pulse 114.

Subsequent Course.—On January 17, Dr. Gayle Crutchfield noted little change in the neurologic picture. The right prepatellar reflex was somewhat more active but he was still unable to elicit other deep reflexes in the lower extremities.

Oscillometer readings by Dr. Nathan Bloom, January 22, were as follows:

	Right Leg	Left Leg
Midthigh.....	0.5 to 1 unit	0.5 to 1 unit

and on February 10, were:

Midthigh.....	1.5 to 2 units	1.5 units

He was discharged from the hospital. February 14, 1939, two months after admission. His weight, normally, was about 165 pounds but at this time it was only 137 pounds.

On May 25, 1939, his weight was 160 pounds, and his general condition was excellent. Blood pressure 138/85. Heart sounds were normal in character, and the heart was not enlarged. No mass could be felt in the region of the aneurysmal sac. There were neither motor nor sensory disturbances in the extremities. Reflexes were normal. There were pulsations in the femoral, popliteal, and posterior tibial arteries but none could be made out in the dorsalis pedis arteries.

Oscillometer readings by Dr. Nathan Bloom, May 25, 1939, were as follows:

Right Arm (at pressure of 100 Mm.Hg.)
5 units

	Right Leg (at pressure of 90 Mm.Hg.)	Left Leg (at pressure of 90 Mm.Hg.)
Midthigh.....	3.5 units	4.0 units
Below knee....	4.0 units	3.5 units
Ankle.....	2.0 units	2.5 units

The patient was seen again, January 30, 1940. His weight was 165 pounds. He stated that his feet became cold a little more noticeably than they had previously but otherwise he had no trouble in his lower extremities. Abdominal examination showed the incision firmly healed, with no evidence of hernia. The abdomen was flat and easily palpated. A bruit could be made out just to the right of the umbilicus and one and one-half inches above it. The bruit was more pronounced during systole but appeared to be continuous in character, suggesting an arteriovenous fistula. In other words, it was of the same character as at the time of the first operation, before the aorta was occluded, and was more distinct than in May, 1939. This suggested an arteriovenous fistula between some of the vessels arising below the aortic occlusion, probably the inferior mesenteric

artery and vein. Blood pressure in the left arm was 120/60, and in the left thigh 125/80.

A teleoroentgenogram of the chest showed the transverse diameter of the great vessels to be 4.7 cm.; of the heart 10.9 cm.; of the thorax 26.4 cm., a cardiothoracic ratio of 41 per cent. The lungs were clear. The domes of the diaphragm were normal. Anteroposterior and lateral roentgenograms of the lower dorsal and lumbar vertebrae and sacrum showed no erosion of the vertebral bodies. The psoas muscles were well outlined and no evidence of an aneurysmal mass could be made out in any view.

Comment.—Fascia was used to occlude the aorta at the first operation because only temporary occlusion seemed indicated. The heavy silk ligature was left around the aorta to occlude it during the aneurysmorrhaphy in case the strip of fascia no longer produced subtotal occlusion. The iliac arteries were exposed and heavy ligatures (tapes would have been better) passed around them for the control of retrograde bleeding when the sac was opened. In spite of these precautions there was considerable pressure in the aorta presumably from the lumbar and middle sacral arteries which entered the aorta below the point of occlusion.

It is believed that the strip of fascia has now completely disappeared and that the lumen of the aorta is fully restored. There is no evidence of recurrence of the aneurysm.

SUMMARY

An attempt has been made to collect the cases of aneurysm of the abdominal aorta and common iliac arteries treated by operations (excluding wiring) upon the aorta. The various surgical procedures which may be applicable to these aneurysms (excluding wiring) are discussed.

Two new cases are reported:

Case 1, a poor surgical risk, developed left-sided heart failure with pulmonary edema and died following occlusion of the aorta proximal to the aneurysm.

Case 2, a young man with a ruptured traumatic aneurysm, had a preliminary occlusion of the aorta proximal to the aneurysm and one month later a restorative endo-aneurysmorrhaphy. When examined one year after the endo-aneurysmorrhaphy, the patient appeared to be well; there was no evidence of aneurysm and the lumen of the aorta was obviously patent.

We realize that there is a marked difference between traumatic and spontaneous aneurysms and that the methods of treatment used in one may not be applicable in the other. For example, it is unlikely that one would find a spontaneous aortic aneurysm suitable for the type of operation, reconstructive endo-aneurysmorrhaphy, used in our second case but it seems likely that a small number of spontaneous aneurysms will be found suitable for obliterative endo-aneurysmorrhaphy. Such operations probably should not be attempted unless the aneurysm arises distal to the renal arteries and, almost certainly, should not be attempted when the aorta is diffusely calcified. Proximal occlusion of the aorta should be undertaken as a preliminary operation. This brings about shrinkage of the sac so that at the second operation either the aorta or the common iliac arteries may be ligated immediately distal to the aneurysm.

If the iliac arteries are permanently occluded, care should be taken to see that the ligatures are placed on the common iliacs, not the external iliacs, and the internal iliacs (hypogastrics) should be carefully protected, because of their great importance as collateral channels.

Also, all vessels communicating with the sac should be ligated, insofar as possible, before the sac is opened. Only by the employment of meticulous preliminary preparation can one hope for success in such cases.

Aneurysms of the proximal portion of the abdominal aorta which have such essential arteries as the celiac, superior mesenteric, or both renals arising from the sac, probably should not be treated surgically, while in those aneurysms arising above the renal arteries but without any of the essential arteries originating from the sac, proximal ligation may be justifiable.

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ANEURYSM OF THE ABDOMINAL AORTA*

TREATMENT BY LIGATION

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"ON Sunday, the 12th ult., Mr. James, one of the surgeons of this institution, placed a *ligature* upon the aorta, in a case of *aneurysm* of the external iliac artery. In one of the Exeter papers, it is stated that the operation was '*successfully* performed'; but it was that kind of success of which the Irishman boasted when he had killed his hog, for the patient survived the infliction of the knife only two or three hours. It is an appalling operation, and we hope not to hear of its repetition—at least in a case of *aneurysm of the external iliac artery* (Lancet, **2**, 607, 1828–1829)."

Reports of ligation of the abdominal aorta are rare. Only 24 recorded instances of this operation have been found in the literature; the first by Sir Astley Cooper, in 1817. No doubt others have been performed, but probably without success. In only ten instances was ligation (either complete or partial) performed for aneurysm of the aorta, but was more often undertaken because of aneurysm of the iliac or femoral vessels,[10] gunshot wound of the aorta or its branches,[2] or tumors.[2] In two instances, ligation was distal to the seat of the disease, and in 22 proximal to it. In ten cases, the occlusion was complete and in 14, the vessel was purposely only partially occluded at the time of the operation or became so soon afterwards. In only five instances can the procedure be said to have been successful, as judged by the period of survival and the relief of symptoms. The rather tart report of Mr. James' operation in the Lancet would, therefore, seem to be supported by the experience of others.

Vaughan's patient, upon whom a partial, distal ligation was performed, survived for two years, and was to some extent relieved of symptoms. Watts' patient lived three and one-half years, and LaRoque's was living 14 months after operation. In Matas' patient, the occlusion was not complete, and she survived with relief for a year and five months. Brooks completely occluded the aorta for aneurysm, and his patient lived for three months. It is unfortunate that these last two patients died of causes unrelated to the disease or the operation, namely, one from tuberculosis, and the other from intestinal obstruction. Most often, death resulted from shock and hemorrhage within a few hours. However, nine patients were operated upon before the days of listerism, and the first four, without the aid of an anesthetic. Progressively better results give promise of its successful accomplishment more often in the future.

The 24 previously reported cases are reviewed in outline in the accompanying table and the procedure in each instance is diagrammatically illustrated. To these is added another operated upon by me.

* Read before the American Surgical Association, St. Louis, Mo., May 1, 2, 3, 1940.

TABLE I

LIGATIONS OF THE ABDOMINAL AORTA

No.	Operator	Date	Publication	Indication	Result	Comment
1	Cooper[1]	1817	Lectures on Surgery. Boston, Wells and Lilly, 1825	L. iliofemoral aneurysm	Lived 40 hrs.	Ligation just above bifurcation. At autopsy, aorta sealed with cloth 1 inch above ligature
2	James[2]	1825	Med. Chir. Tr., **16**, 1830	L. ext. iliac aneurysm	Lived 3 hrs.	Complete ligation just above bifurcation. Pain and deadness in legs
3	Murray[3]	1834	London Med. Gaz. **14**, 68, 1834	R. iliofemoral aneurysm	Lived 23 hrs.	Complete ligation just above bifurcation
4	Monteiro[4]	1842	Lancet, **1**, 334, 1842–1843	R. iliofemoral aneurysm	Lived 10 days. Death from hemorrhage—due to infection	Partial occlusion with silk just above bifurcation. Satisfactory clotting
5	South[5]	1856	Lancet, **2**, 47, 1856	R. common and ext. iliac aneurysm	Lived 43 hrs.	Ligation at bifurcation. First operation performed under an anesthetic
6	McGuire[6]	1868	Am. Jour. Med. Sci., **56**, 415, 1868	Aortic aneurysm extending into both iliacs	Lived 11 hrs.	Ligation just below inferior mesenteric artery. Rupture of sac required aortic ligation
7	Stokes[7]	1869	Dublin Quart. Jour. Med. Sci., **48**, 1, 1869	R. iliofemoral aneurysm	Lived 13 hrs. Death from shock	Complete ligation with silver wire at bifurcation. Contents of sac clotted. Collateral circulation good
8	Watson[8]	1869	Brit. Med. Jour., **2**, 216, 1869	Iliac aneurysm	Lived 65 hrs.	Common iliac tied 9 wks. previously. Aorta ligated with silk at bifurcation for secondary hemorrhage; ext. and int. iliacs also ligated
9	Czerny[9]	1870	Wien. med. Wchnschr., **1**, 402, 1870	Wound of left femoral artery	Lived 27 hrs. Death from sepsis and hemorrhage	Because of bleeding—aorta ligated by mistake for common iliac. Circulation good on right
10	Czerny[10]	1879	Centralbl. f. Chir., **6**, 737, 1879	Tumor of left kidney	Lived 10 hrs.	Severe hemorrhage from renal artery necessitated ligation of aorta and left renal artery
11	Milton[11]	1890	Lancet, **1**, 85, 1891	Ruptured aortic aneurysm	Lived 24 hrs. Death from hemorrhage and shock	Ligation with silk below renal vessels for ruptured aneurysm
12	Keen[12]	1899	Am. Jour. Med. Sci., **120**, 251, 1900	Aortic aneurysm	Lived 48 days. Death from cutting of ligature	Partial ligation just below diaphragm. Gradual reduction in size

12	Keen[12]	1899	Am. Jour. Med. Sci., **120,** 251, 1900	Aortic aneurysm	Lived 48 days. Death from cutting of ligature	Partial ligation just below diaphragm. Gradual reduction in size
13	Körte[13]	1899	Deutsche med. Wchnschr., **26,** 717, 1900	R. common iliac aneurysm	Lived 1 hr.	Common iliac ligated 37 days previously. Aorta ligated with silk at bifurcation. Death from hemorrhage
14	Tillaux[14]	1900	Bull. et Mém. Soc. de Chir. de Paris, 1900	L. iliac aneurysm	Lived 39 days. Femoral vein thrombosed	Partial ligation; ligature slipped. Aorta ligated by mistake for common iliac
15	Morris[15]	1901	ANNALS OF SURGERY, **35,** 207, 1902	Aortic aneurysm extending from celiac axis to mesenteric vessels	Lived 53 hrs. Gangrene of intestine from pressure of clamps	Partial distal ligation by catheter held with clamps
16	Scott[16]	1905	Hosp. Bull. Univ. Maryland, **1,** 41, 1905	Gunshot wound of adominal aorta	Died—1 hr.	Ligation with catheter and forceps just above bifurcation
17	Halsted[17]	1906	Surgical Papers, **1,** 321, 1924	Aneurysm of abdominal aorta	Death—18 days after second band. Rupture intrathoracic	Band applied to thoracic aorta. 23 days later, band applied to abdominal aorta distal to aneurysm
18	Halsted[18]	1909	Surgical Papers, **1,** 321, 1924	Aneurysm of abdominal aorta	Death from infection—47 days after band was placed	Band applied between renal and sup. mesenteric arteries. Later wired
19	Hamann[19]	1917	ANNALS OF SURGERY, **68,** 217, 1918	Tumor of pelvis	Lived 6 mos. Death from hemorrhage from bed sore	Ligation just above bifurcation. At autopsy, aorta only partially occluded
20	Vaughan[20]	1920	ANNALS OF SURGERY, **74,** 308, 1921; also **76,** 519, 1922	Aneurysm of abdominal aorta at origin of sup. mesenteric artery	Lived 2 yrs. and 1 mo.	Distal ligation with cotton tape. Apparently improved during life. Autopsy showed partial occlusion
21	Watts[21]	1923	Trans. South. Surg. Assoc., **43,** 245, 1931 (quoted by LaRoque[24])	R. common iliac aneurysm	Lived 3½ yrs.	Partial ligation with tape just below sup. mesenteric artery. Improvement. Death due to rupture
22	Matas[22]	1925	ANNALS OF SURGERY, **81,** 457, 1925	Aneurysm of abdominal aorta at bifurcation	Lived 1 yr. 5 mos. Died of tuberculosis	Double complete ligation, with cotton tape, at bifurcation. Sac clotted but not completely occluded. First successful ligation
23	Brooks[23]	1926	J.A.M.A., **87,** 722, 1926	Aneurysm of abdominal aorta at bifurcation	Lived 3 mos. Died intestinal obstruction	Complete ligation with fascia and silk just above bifurcation. Apparently cured
24	LaRoque[24]	1929	Trans. South. Surg., Assoc., **43,** 245, 1931	R. common iliac aneurysm	Living 14 mos. after operation	Partial ligation with silk and cotton tape at bifurcation
25	Elkin[25]	1939	ANNALS OF SURGERY, **112,** 895, 1940.	Aneurysm of abdominal aorta at bifurcation	Alive 11 mos. after operation. Improved	Double partial ligation with tape proximal to aneursym. Reduction in size. Bruit present

Case Report.—Hosp. No. 90842: J. E. C., white, male, age 61, a minister, was admitted to the Emory University Hospital, May 28, 1939, complaining of a tumor in his abdomen and of pain in his legs. His past health had always been good. His mother, father and two brothers died of cardiovascular disease.

About six months previously, he had noticed a swelling in the left side of his abdomen but paid little attention to it since it was not causing pain. The mass grew progressively larger, and the pain, which was at first intermittent, became constant, and was of such severity as to keep him awake. The pain extended over his lower abdomen and into his

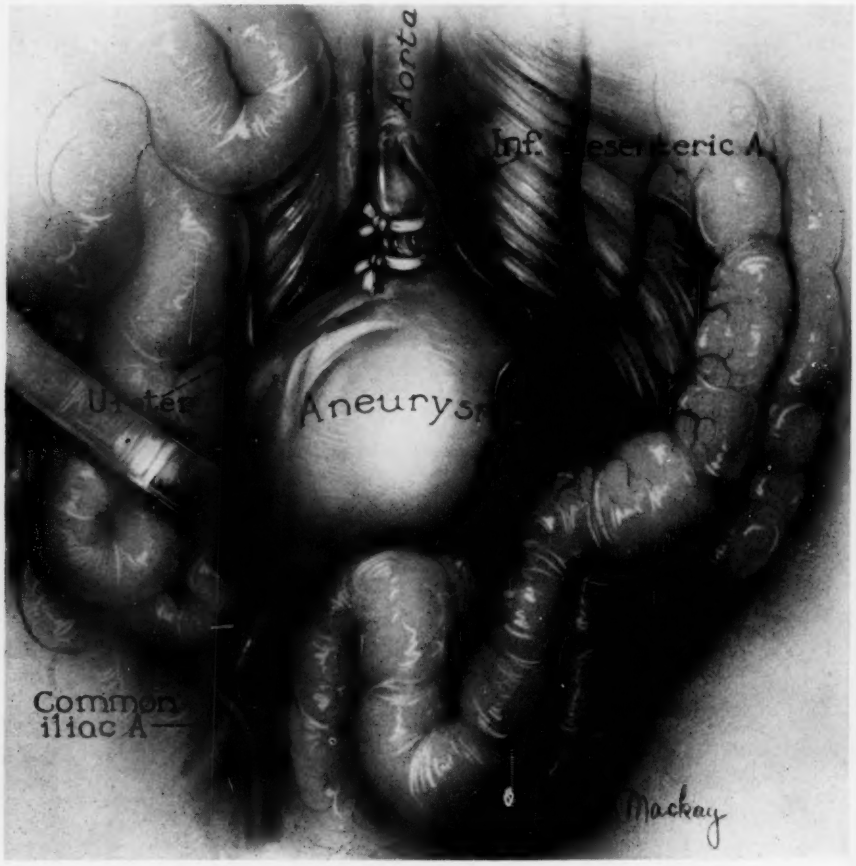


FIG. 1.—The appearance of the aneurysm at operation. Double ligation, with cotton tape, between the inferior mesenteric artery and the bifurcation.

legs. He occasionally had severe "cramps" in the calves of his legs. His feet were colder than normal, and numbness and tingling were frequently experienced. During the month preceding examination, he had noticed that the abdominal mass pulsated.

Physical Examination.—There was a marked thickening and tortuosity of his peripheral vessels. His heart was normal in size. There were occasional extrasystoles. In the lower abdomen, a rounded mass could easily be felt. It was about five inches in diameter with its upper margin at the level of the umbilicus. It pulsated synchronously with the heart beat. There was no thrill and no bruit. The circulation in his legs was good; the femoral, popliteal, dorsalis pedis and posterior tibial vessels were pulsating. His blood pressure at the cubital fossa was 140 systolic and 100 diastolic. The pressure in the popliteal vessels was approximately the same.

Roentgenologic examinations showed arteriosclerosis of the aorta and the peripheral vessels. There was some calcification in the abdominal mass, which could be seen pulsating under the fluoroscope. The Wassermann and Kahn reactions were negative.

Operation.—June 1, 1939: Under cyclopropane and ether anesthesia. The abdomen was opened through a left rectus incision. The aneurysm was immediately disclosed. It occupied the lower aorta from a point just below the inferior mesenteric artery to the bifurcation. The dilatation extended slightly into both common iliac arteries. Calcified plaques could be felt in the aneurysm and in the aorta above it. The posterior peritoneum was opened through an avascular area, and the aorta was dissected free of its surrounding tissues at a point just below the origin of the inferior mesenteric artery. This was difficult because of the calcification of the vessel and its adherence to the surrounding tissues. When the aorta was completely isolated, it was partially ligated at two places with one-fourth inch cotton tape (Fig. 1). The ligatures were placed about one-half inch apart. The tapes were tied until the pulsation in the aneurysm was almost completely obliterated and until the pulsation of the femoral vessels could scarcely be felt. Since there was no increased vascularity about the aneurysm and no evidence of a compensatory collateral circulation, it was thought best to only partially occlude the circulation to the extremities. Moreover, the marked arteriosclerosis of the peripheral vessels seemed to make complete ligation unwise. The peritoneum was closed over the ligatures and the abdomen closed in layers.

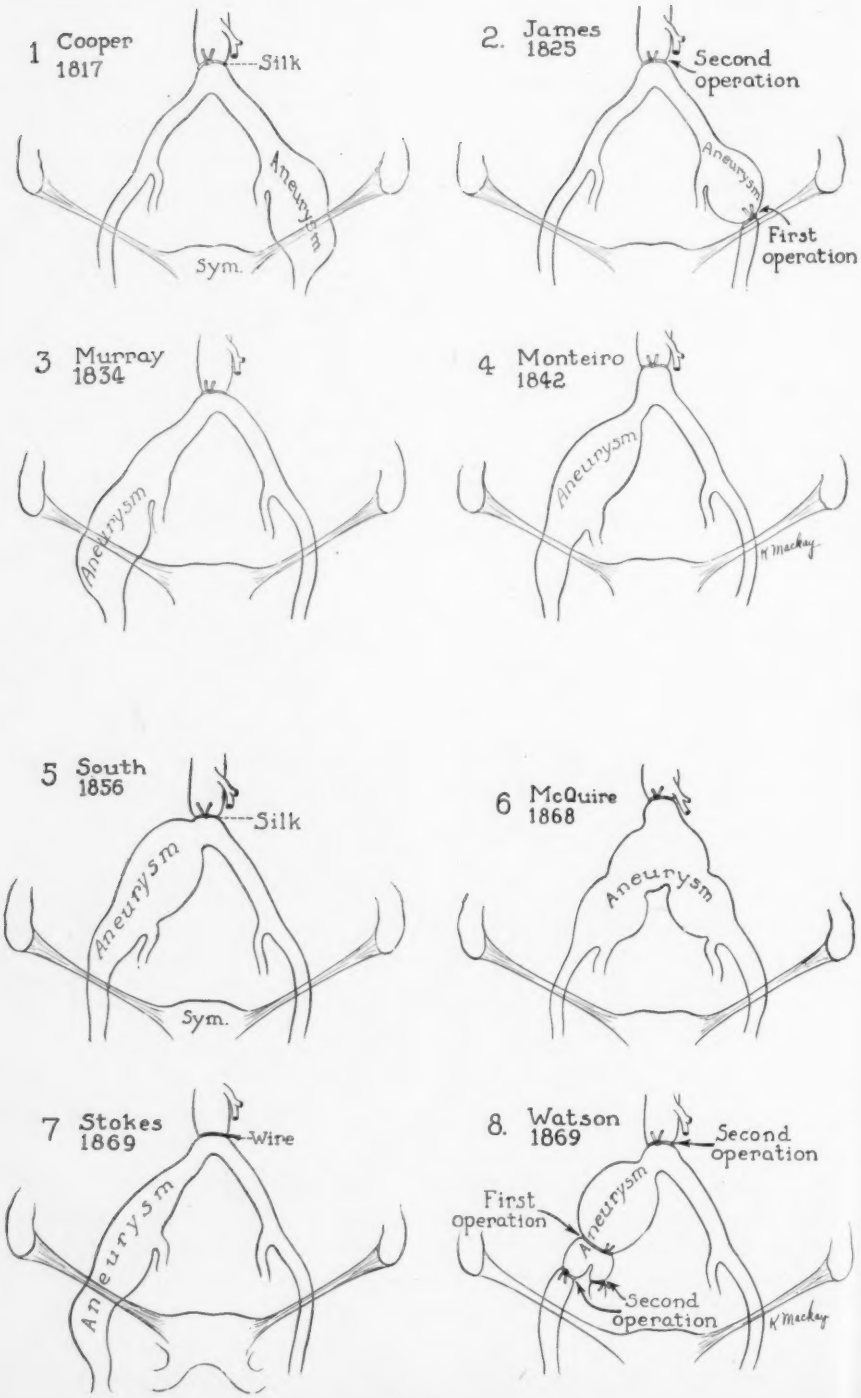
Postoperative Course.—There was no remarkable change in the heart sounds following this ligation. The systolic blood pressure gradually rose during the operation from 140 to 180 and the diastolic from 90 to 100. The rate of the pulse and respirations was not remarkably affected (Fig. 2). Following operation, he complained of some coldness in his legs but this disappeared in two days. The systolic blood pressure in the popliteal vessels dropped to 60 Mm.Hg. Within a week, he was completely relieved of pain in his abdomen and legs. He was allowed out of bed on the twelfth day and returned home, two weeks after operation. At this time, the mass was considerably smaller and could be felt only as an area of induration in the left lower quadrant. There was no pulsation, no thrill, and no bruit. Within a month he resumed his duties as a country minister and since that time has been actively engaged in his work. He drives a car daily without difficulty.

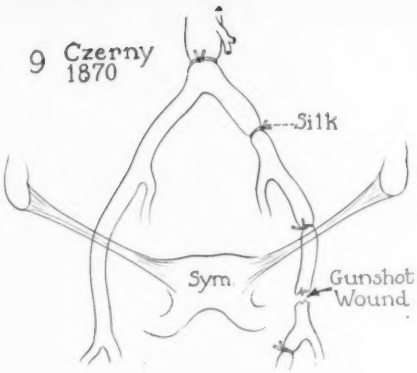
Six months later the mass could be felt but was much smaller than originally. There was a slight pulsation in it and a definite systolic bruit could be heard over it. Since that time, there has been no change in the mass and, now, 11 months after operation, he is free from pain and able to carry out his duties. The pulsation in the femoral artery is forceful, but the pulsation in the dorsalis pedis and posterior tibial arteries is weak and inconstant. It would appear that the partial ligation has so slowed the current of blood through the aneurysm as to bring about partial clotting but complete occlusion has not been accomplished.

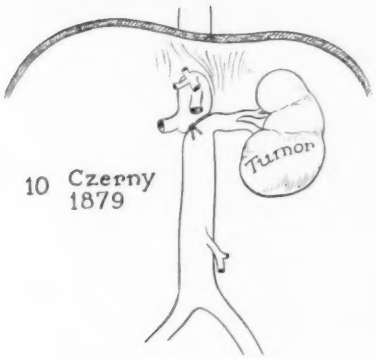
There are certain inherent difficulties in ligation of the aorta aside from its technical performance. This probably accounts for the small number of attempts to occlude the vessel.

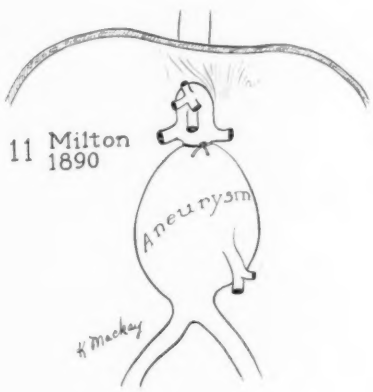
(1) *The location of the aneurysm* is rarely at a point where proximal ligation can be undertaken without menace to the vitality of the kidneys or intestine. The most frequent site is in the region of the celiac axis, and partial distal ligation is usually the only procedure that can be carried out. Theoretically, this may slow the blood current and produce clotting and delay rupture, as in Vaughan's case, but, practically, it is rarely successful.

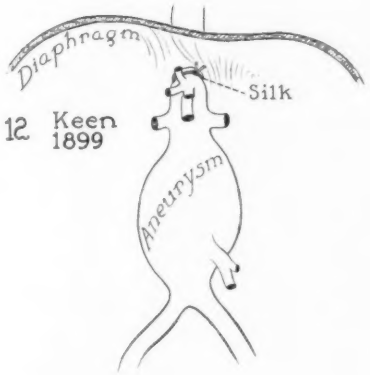
Only rarely is an unruptured aneurysm found at the bifurcation and in such a position that the aorta can be occluded distal to the inferior mesenteric

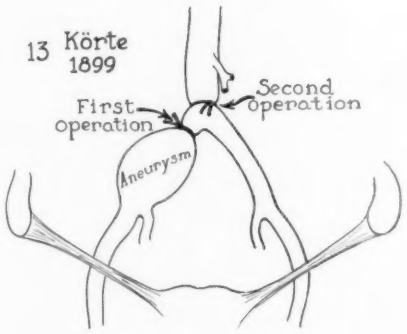

1 Cooper 1817
Silk
Aneurysm
Sym.
2. James 1825
Second operation
Aneurysm
First operation
3 Murray 1834
Aneurysm
4 Monteiro 1842
Aneurysm
H Mackay
5 South 1856
Silk
Aneurysm
Sym.
6 McQuire 1868
Aneurysm
7 Stokes 1869
Wire
Aneurysm
8. Watson 1869
Second operation
First operation
Aneurysm
Second operation
K Mackay

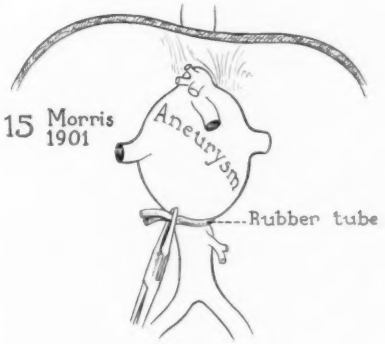

9 Czerny
1870
Silk
Sym
Gunshot
Wound

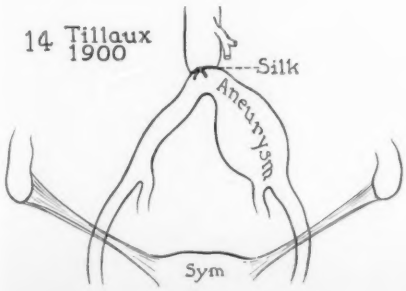

10 Czerny
1879
Tumor

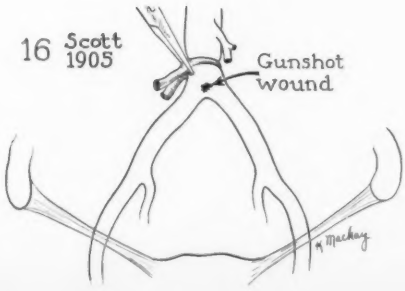

11 Milton
1890
Aneurysm
H. Mackay

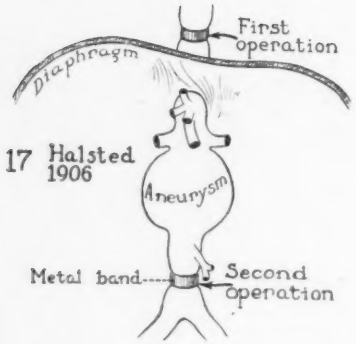

Diaphragm
Silk
12 Keen
1899
Aneurysm

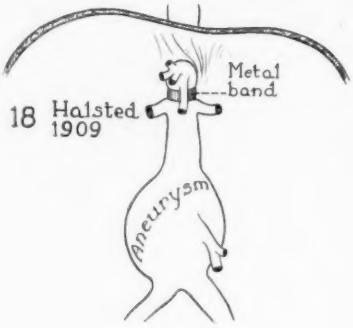

13 Körte
1899
First
operation
Second
operation
Aneurysm

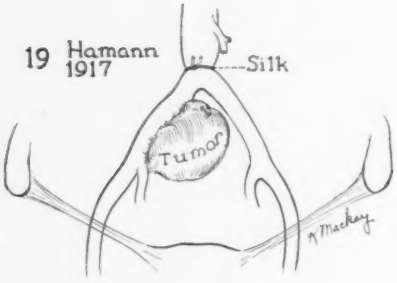

15 Morris
1901
Aneurysm
Rubber tube

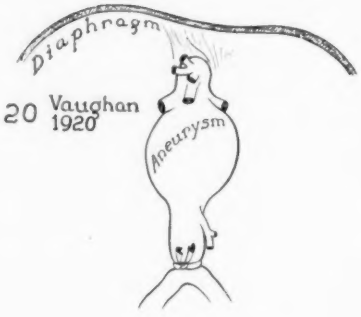

14 Tillaux
1900
Silk
Aneurysm
Sym

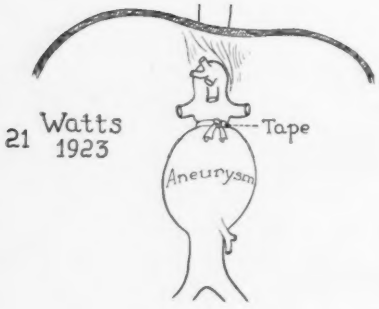

16 Scott
1905
Gunshot
wound
H. Mackay

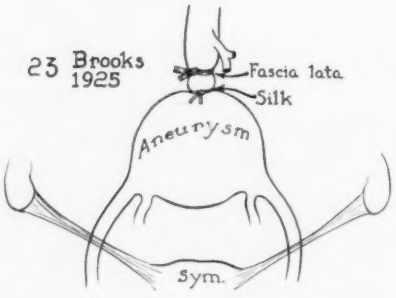

First operation
Diaphragm
17 Halsted 1906
Aneurysm
Metal band
Second operation

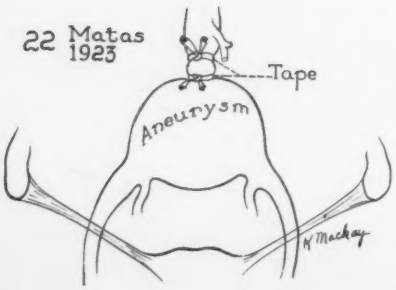

Metal band
18 Halsted 1909
Aneurysm

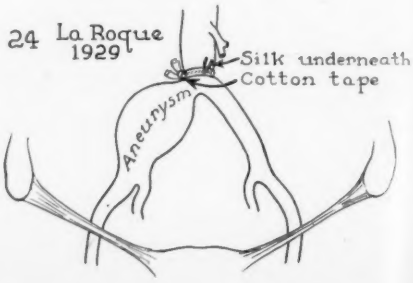

19 Hamann 1917
Silk
Tumor


Diaphragm
20 Vaughan 1920
Aneurysm


21 Watts 1923
Tape
Aneurysm

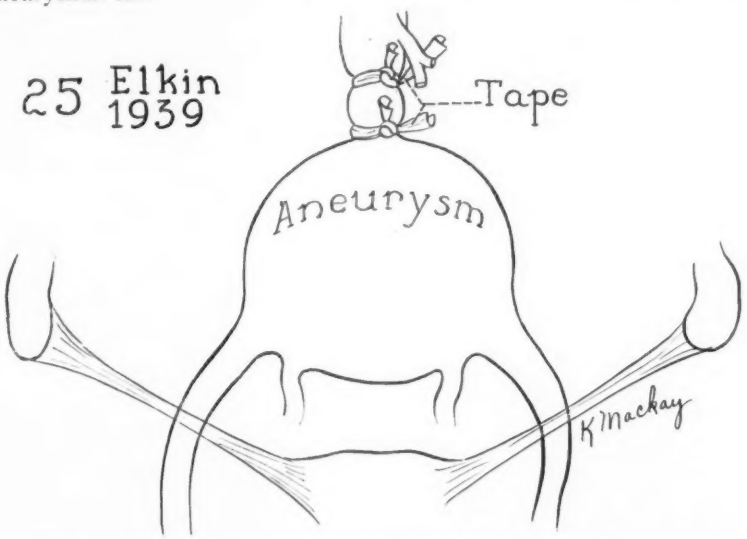

23 Brooks 1925
Fascia lata
Silk
Aneurysm
Sym.


22 Matas 1923
Tape
Aneurysm


24 La Roque 1929
Silk underneath
Cotton tape
Aneurysm

artery. Complete ligation above that vessel would, very likely, result in gangrene of the sigmoid.

(2) *An efficient collateral circulation* rarely exists about an aortic aneurysm, and gangrene of the extremities will result if the vessel is occluded. In only one instance (Brooks[23]) had the communications developed sufficiently to allow the surgeon to completely occlude the vessel. Moreover, this same collateral supply may be the cause of an early recurrence as vessels reenter the aneurysmal sac.



(3) *The type of ligature* and *the manner of occlusion* still remain an unsolved problem. Partial occlusion with metal bands (Halsted[17, 18]) or with silk or wire is usually unsuccessful because of the danger of rupture of the vessel at the point of ligature. Bands of fascia or tape frequently give way and allow the channel to reopen. Partial double ligation plus the introduction of some sclerosing substance may overcome these disadvantages.

(4) *The effect on the heart* has been the subject of considerable dispute. Kast,[26] in 1880, concluded, from a series of experiments, that the blood pressure in the aorta did not rise proximal to the point of ligation. It was his opinion that the effect on the heart was not dangerous to life. However, Katzenstein,[27] in 1905, formed exactly the opposite opinion since he found an elevation in the blood pressure of the artery proximal to the site of the occlusion and dilatation and hypertrophy of the heart. Matas[22] believed that total occlusion of the abdominal aorta imposed a strain upon the heart. In Brooks' case the heart was found to be essentially normal three months after the ligation of the aorta, but in this instance a considerable collateral circulation was already established.

Brooks, Blalock and Johnson[28] found, following a series of experiments in dogs, that the cardiac output was decreased and there was little, if any, change in the blood pressure proximal to the occlusion. They found no evidence of hypertrophy of the heart.

In the case here reported, there was a gradual rise in the blood pressure throughout the operation. A continuation in this rise following partial occlusion of the aorta cannot, therefore, be attributed to the occlusion of the vessel (Chart 1).

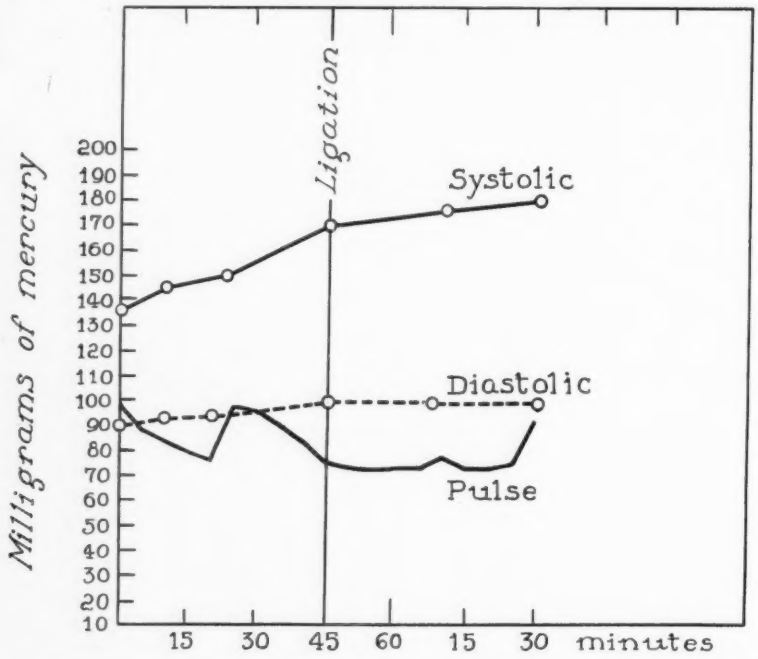


CHART 1.—Showing the systolic and diastolic blood pressure and the pulse during operation. The rise in blood pressure was gradual throughout and, therefore, could not be attributed to the ligation of the aorta.

Recently, the opportunity to observe the effect in another patient was offered (Chart 2). In this case the aortic aneurysm in the region of the celiac axis was treated by distal ligation just above bifurcation. There was no change in the blood pressure for 15 minutes. After 15 minutes there was a sudden drop of systolic pressure from 120 to 80 with evidence of circulatory failure. Following stimulation and a lowering of the patient's head the blood pressure rose, in about 30 minutes, back to 120 and was maintained at this level until rupture of the aneurysm produced death two days later. It was thought that this sudden fall in blood pressure was due to cardiac failure but why this should occur without previous rise in arterial pressure is not understood.

DISCUSSION.—This report is concerned primarily with the effects of ligation of the abdominal aorta and consideration of those cases in which this procedure has been carried out. Therefore, the treatment of aortic aneurysm by other methods has not been considered. In only six patients, upon whom ligation has been performed, may the procedure be considered in any degree successful. Other means of treating an aneurysm of this vessel should be considered, but it is improbable that wiring, coagulation, or the application of the Matas principle of endo-aneurysmorrhaphy could be carried out with any great hope of

success. The effect upon the heart and circulation should be further studied and methods of producing occlusion by other means than ligation must be developed before the operation can be successfully performed in the majority of cases.

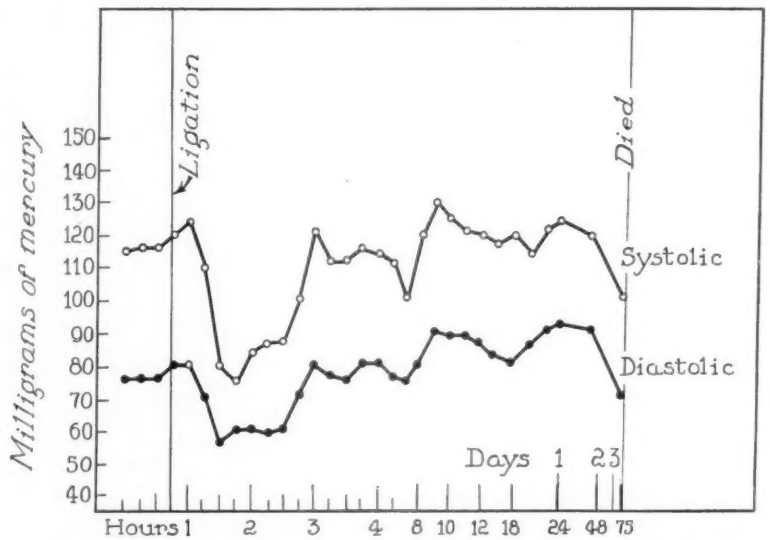


CHART 2.—Showing the systolic and diastolic pressure in a patient upon whom the aorta was ligated distal to the aneurysm. Note the marked drop in blood pressure after ligation. This was attributed to cardiac failure.

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DISCUSSION.—DR. I. A. BIGGER (Richmond, Va.): I wish to congratulate Doctor Elkin on his excellent presentation of this interesting case. In this discussion, I would like to emphasize certain points regarding the decision as to whether or not ligation should be attempted in patients with abdominal aortic aneurysms. If one of the essential vessels, such as the superior mesenteric, comes off from the aneurysmal sac, it seems obvious that no curative procedure should be attempted. In other words, it would seem to me that complete proximal occlusion of the aorta in the presence of an aneurysm involving the origin of one of these vessels is worse than useless, for obliteration of the sac by clot will produce occlusion of the superior mesenteric or the celiac arteries, which will almost certainly prove fatal.

There is some doubt, I think, as to whether a proximal ligation can be considered more than a palliative procedure in the great majority of cases of aneurysm, although there have been, as pointed out by Doctor Elkin, some instances of apparent cure by proximal ligation alone. In spite of this, it seems likely that the incidence of cure of aortic aneurysm from proximal ligation will be small. Because of this impression, we were not content with proximal ligation in the following case.

About 18 months ago we operated upon a man, age 25, for a ruptured traumatic aneurysm of the abdominal aorta. At the first operation we occluded the aorta proximal to the aneurysm and above the inferior mesenteric artery. It has been shown that occlusion of the inferior mesenteric artery is not likely to be followed by serious interference with the circulation to the segment of bowel supplied by that vessel. After one month we again operated upon this patient and performed an endo-aneurysmorrhaphy.

Following occlusion of the aorta, this man developed complete paralysis of his lower extremities, which persisted for about two weeks and then gradually improved. He also had marked sensory changes which cleared up after about six weeks and he now has excellent function in his lower extremities.

A heavy strip of fascia lata was used for the occlusion, for the reason that we had in mind to attempt endo-aneurysmorrhaphy, and we felt that it would be desirable to have a reestablishment of the circulation through the aorta after the sac had been obliterated. This has occurred, and he now has normal pulsations in all of the vessels of the lower extremities. No evidence of recurrence of the aneurysm can be found.

I realize, of course, that a traumatic aneurysm in a young man is an entirely different problem from that presented by spontaneous aneurysms which usually occur in more elderly people, but I do believe that there are some patients with spontaneous aneurysms of the distal portion of the aorta in whom aneurysmorrhaphy might be successful, if undertaken in stages.

If such an operation is to be attempted, it would seem wise to perform a preliminary subtotal occlusion of the aorta proximal to the aneurysm. After a reasonable time, say a month or six weeks, when the sac has become shrunken, an occlusion of the vessel or vessels distal to the aneurysm can be more readily accomplished. After the collateral circulation has been controlled, in so far as possible, some modification of the Matas operation should be performed. This should offer a greater chance of cure than proximal ligation alone.

In spontaneous aneurysms, restorative aneurysmorrhaphy would not be possible, so tape, or similar material, should be used to produce permanent occlusion of the aorta both proximal and distal to the sac.

Dr. Rudolph Matas (New Orleans, La.): As a pioneer in this field, I heartily congratulate Doctors Elkin and Bigger on their successful operation for the cure of abdominal aneurysms. They have brought a note of cheer and encouragement to the classically somber tone of aortic surgery.

Taken together with Doctor Owings' experimental success in the segmental obliteration of the lower thoracic and abdominal aortic tract by his method of gradual occlusion, these actual clinical achievements would suggest that surgery is gradually approaching some mastery over one of its most rebellious provinces.

Doctor Elkin's case appeals to me particularly, because his experience duplicates and largely confirms the value of the technic adopted in the successful ligation of the aorta for an aneurysm of the bifurcation and iliacs which I reported to the Association at Baltimore, in 1924. In that case, the occlusion was complete when applied, but became partial on the ninth day, when the pulsation returned in the sac, showing that the ligatures had yielded sufficiently to allow a small stream to flow through a narrow channel in the ligated segment. While this reduced stream gave great relief to the cardiopulmonary circulation and no doubt saved the patient's life, it did not prevent the ultimate cure of the aneurysm, because this had been completely consolidated by laminated clot and had ceased to pulsate long before her death, which occurred one year and five months after the ligation, consequent to an overwhelming hemorrhage from a tuberculous cavity in the lung.

Doctor Elkin used two cotton tapes, as I did, and, I presume, from his patient's excellent recovery, they were as well tolerated by the aorta of his patient. For, as I will show later, the histology of the ligated segment demonstrated that, contrary to the usual experience, the tapes had not cut through but had been completely incorporated in the walls of the artery without injuring the intima in the least. The ligated segment was entirely covered by a capsule of dense connective tissue which blended perfectly with an external

coat of the aorta, greatly strengthening the artery while completely concealing the ligatures. Judging by the histologic study of the ligated segment of the aorta in my patient's case and by Doctor Elkin's success, cotton tape, which was first used to ligate the aorta by Dr. G. T. Vaughan, of Washington, at Doctor Halsted's suggestion (with survival of more than two years), is a dependable material and, in fact, is so well tolerated and assimilated by the aortic walls that it would seem to make the use of autogenous fascial and other organic tissue material a thoroughly unnecessary and superfluous performance. Our experience with cotton tape would seem to bear out the general conclusions of Meade and Ochsner (1940) in making cotton a preferable material for buried sutures and ligatures.

I am very much obliged to Doctor Elkin for the pains he has taken to illustrate the procedure adopted in my patient's case. His drawings will serve as an appropriate introduction to the lantern slides exhibit that is to follow this discussion.

Doctor Bigger's operation is also of special interest to me as it is the first successful application to the aorta of my method of intrasaccular suture (endoaneurysmorrhaphy) for the cure of aneurysm. We have records of five endoaneurysmorrhaphies for abdominal aortic aneurysms (R. Lozano, 1905; J. C. Munro, 1906; G. W. Crile, 1907; J. H. Gibbon, 1912), all of which ended fatally, except the last operation performed by Doctor Bigger, January 10, 1939. All of these operations were performed under desperate and seemingly hopeless circumstances for ruptured or leaking sacs, two dying on the table and the two others surviving only a few hours. Doctor Bigger's patient is the only traumatic aneurysm caused by gunshot wound. The history of this complicating injury, as related by Doctor Bigger, is a fine example of the resourcefulness of contemporary surgery under good generalship, especially since unlimited blood transfusion has become so universally available, besides the benefits of autotransfusion, which was a feature of this difficult case.

In addition to the five abdominal aneurysmorrhaphies, there are two cases of thoracic aneurysms in which the method of suture has been resorted to: One, a traumatic aneurysm of the descending thoracic aorta on which Kummel, of Hamburg, performed a constructive aneurysmorrhaphy (1914), technically a fine success, but unfortunately in a hopeless condition. The other is a sacculated aneurysm of the ascending arch of the aorta, reported by V. V. Kerstorsky (1927), in which the sac was torn in the course of an intrathoracic exploration. An immense hemorrhage flooded the field, which was controlled by clamping the broad pedicle of the sac, thereby excluding it from the arch of the aorta, the exclusion of the sac being completed by a series of continued mattress sutures. Hemostasis was secured, but the patient was so depleted by hemorrhage and shock that he died on the table before he could be transfused.

Other cases could be quoted from the recent literature which indicate that thoracic surgeons, emboldened by the great progress in the technic of pulmonary surgery, are making more frequent attempts to explore for aneurysm of the aorta and heart, in the hope that a sacculated aneurysm may be found in its early stages which will lend itself to conservative methods of suture. Thus far, the great majority of all these attempts in the thorax have proved abortive, usually ending with an exploration or with some heroic episode which has not encouraged many renewals of the same experience.

We may safely assert that, for the present, at least, the hope of aneurysmal therapeutics in the chest must depend much more on prophylaxis than on surgery, including the recently attempted resurrection of the methods of wiring and electrolysis.

ANEURYSM OF THE ABDOMINAL AORTA AT ITS BIFURCATION INTO THE COMMON ILIAC ARTERIES*

A PICTORIAL SUPPLEMENT ILLUSTRATING THE HISTORY OF CORINNE D., PREVIOUSLY REPORTED AS THE FIRST RECORDED INSTANCE OF CURE OF AN ANEURYSM OF THE ABDOMINAL AORTA BY LIGATION

RUDOLPH MATAS, M.D.

NEW ORLEANS, LA.

THE HISTORY of this aneurysm, and of the ligation of the abdominal aorta performed for its cure, was originally presented, as a preliminary report, before the American Surgical Association at its meeting in Baltimore, April 18, 1924 (Tr. Am. Surg. Assn., **42**, 603–616, 1924), and in the ANNALS OF SURGERY, **81**, 457–464, February, 1925.

The last report closed with a statement that a final, illustrated report of the case, in all its bearings, would appear in a later publication. Unfortunately, a number of circumstances delayed the completion of the final report, but out of the abundance of the illustrative material which had collected about this patient, a moving picture was made which showed the patient, the aneurysm, the technic of the ligature, and all the gross anatomic and histologic changes in the artery at the site of the ligation. This film was exhibited by the writer at the Dallas meeting (surgical section) of the American Medical Association in April, 1926, at the discussion of Dr. Barney Brooks' report of a successful aortic ligation for a similar aneurysm. This demonstration passed in silence, and the film was not included in the published proceedings of the meeting. Through some unaccountable accident, the film was destroyed or lost while in storage at the promoters' laboratories. However, the original drawings, photographs and other illustrations were, fortunately, preserved in the library of the Medical School (Tulane), and it is from this source that the film exhibited by the writer at the St. Louis discussion of Doctors Elkin's and Bigger's successful ligations was made available for that occasion.

In view of the historic interest attached to the case of the patient, Corinne D., as the first recorded cure of an aneurysm of the abdominal aorta by ligation, and of the fact that the ligatures—contrary to previous experience—were made effective without cutting through the artery or damaging its coats, it would seem that the hitherto unpublished photomicrographs and drawings of the anatomic and histologic changes in the walls of the aorta at the site of its ligation would fit into the discussion of the successful cases of Doctors Elkin and Bigger, if only as an instructive illustration of the gradual evolution of a working technic of aortic surgery in the abdomen, as it was so encouragingly unfolded at this meeting. Besides, and quite apart from any historic interest,

* Introduction to a moving picture of the case of Corinne D., exhibited in the discussion of "Aortic Aneurysms" (Doctors Elkin and Bigger) at the meeting of the American Surgical Association, St. Louis, May 1–3, 1940.

the series of illustrations which are herewith reproduced, will, I trust, help to redeem the promise of their publication made 13 years ago.

SYNOPTIC HISTORY OF THE PATIENT: WITH A RECAPITULATION OF THE MORE SALIENT FACTS AND CONCLUSIONS

Case Report.—*Corinne D.*, colored, female, age 28, field laborer. First admitted to Charity Hospital, (Dr. J. B. Guthrie's clinic), October 25, 1922.

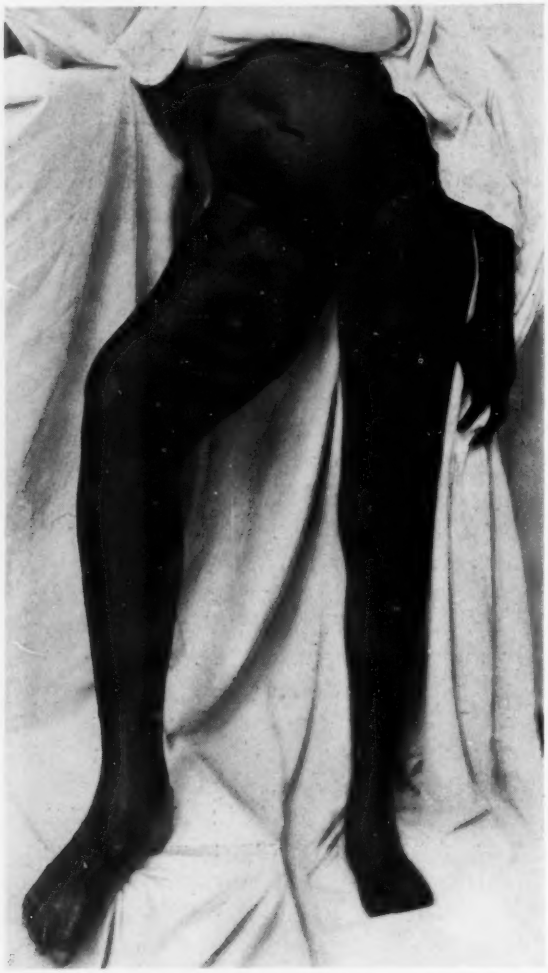


FIG. 1.—Corinne D., age 29. Aneurysm of the common iliac arteries, including the bifurcation of the aorta. Photograph of the patient one year after the ligation of the aorta. Right hip partially ankylosed as a result of arthritis and contracture of the muscles of the foot and leg by edema, due to compression of the anterior crural nerve and right common iliac vein. Good circulation in the upper and lower extremities despite low blood pressure and feeble pulses.

Diagnosis.—Malignant syphilitic infection with generalized manifestations—eight months' duration before admission (fever, acute polyarthropathies and myalgias specially localized in lumbar spine, hip, knee and other joints of the lower extremities; general adenopathy, tibial periosititis and multiple suppurating syphilides on legs, arm and body).

Wassermann strongly positive. A helpless and crippled invalid when admitted. Under vigorous antisyphilitic treatment, was so much improved that, by December 16, 1922, she considered herself well and was discharged at her own request.

Readmitted March 6, 1923 (service of Drs. E. D. Martin and A. C. King). Neglected treatment, and relapsed into a worse condition than on first admission. In addition to the

FIG. 2. FIG. 3. FIG. 4.

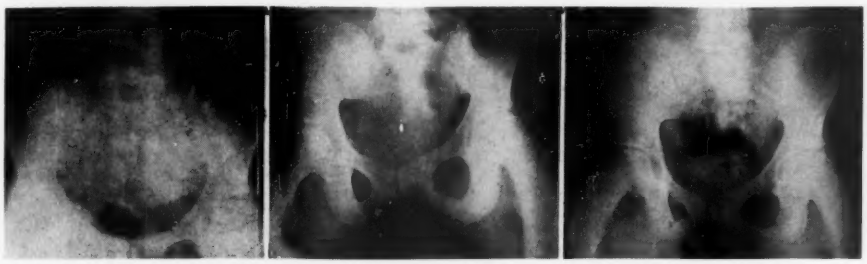
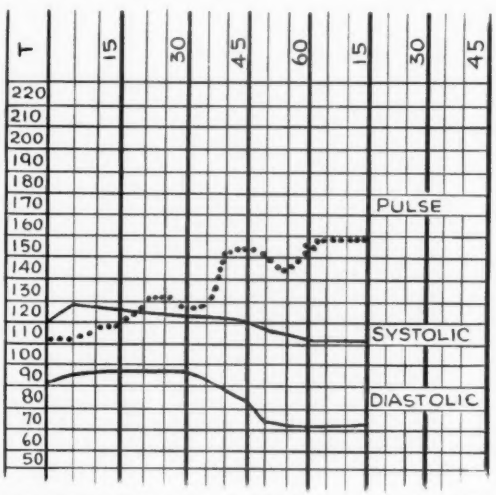


FIG. 2.—Teleradiograph of pelvis, April 4, 1923, five days before the ligation of the aorta.

FIG. 3.—Teleradiograph of pelvis nearly two months after ligation of the aorta. The shadows over the sacro-iliac region extending into the right sacro-sciatic notch and over the sacrum obscure the outlines of the bone. The right half of the pelvic cavity is also obscured by the ill-defined shadow of the sac as it was beginning to fill with clot. The erosion of the left trochanter from syphilitic osteo-periostitis is also plainly shown.

FIG. 4.—Teleradiograph of pelvis, March 27, 1924, nearly one year after the ligation of the aorta showing marked reduction in the aneurysmal shadow.



ANESTHETISTS CHART: Corinne D., ligation of abdominal aorta, 1/9/23. Dr. A'lgeyer anesthetist—Gas-oxygen. Preliminary morph. sulph. gr. ¼ + atropine sulph. gr. 1/150. Anesthesia commenced at 9:08 A.M. Operation commenced 9:28 A.M. Anesthetic discontinued 10:30 A.M.; operation 10:30 A.M. Note rise of pulse and dropping of blood pressure on ligation of the aorta, 45 minutes after beginning of operation, and persistent high pulse rate (155-160) after the ligation, with notable increase in the respiratory rate after the ligation (24-38).

arthropathies which crippled her spine, right hip and other parts of her lower extremities, she had developed an aortic aneurysm (a sequela to an acute aortitis) which involved both common iliacs and the aortic bifurcation. Coincidently with the relapse in the arthropathies of the spine, hip and other parts of the lower extremities and the appearance of the pulsating tumor, a remittent fever had developed ranging from 100° to 104°+ F., which at first suggested a retroperitoneal abscess. This diagnosis was dismissed as the fever gradually subsided, leaving the aneurysmal tumor growing visibly, and rapidly, with increasing lumbar and radiating pressure pains in the pelvis and lower extremities along the right sciatic and anterior crural tracts. By April 3, 1923, the tumor had filled the pelvis,

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FIG. 5.

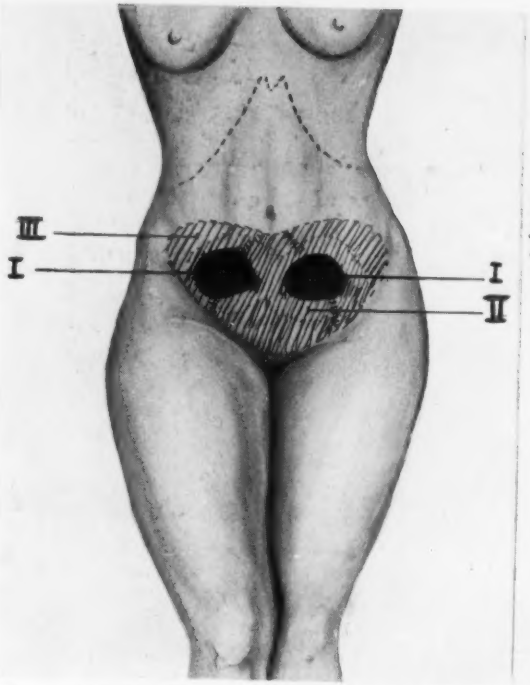

FIG. 6.

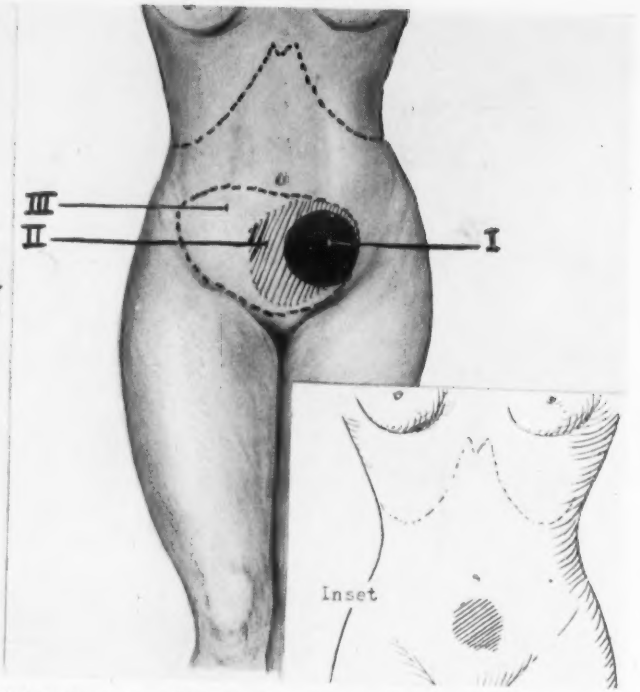

FIG. 5.—Diagrammatic outline of tumor area to show the two centers or vortices of the greatest circulatory activity before the operation.

I. Bilateral centers of loudest murmurs and most vigorous pulsation.
II. Middle zone of moderate pulsation and bruit.
III. Outer zone of least intensity.

FIG. 6.—Diagrammatic outline of contracting area of tumor as felt, April 8, 1924, one year after the ligation of the aorta.

I. Contracting center of pulsation and feeble bruit.
II. Zone of less active pulsation and inaudible bruit.
III. Zone of totally absent pulsation and bruit in gradually shrinking sac.

Inset.—Area of palpable tumor felt, June 14, 1924, two months after the preceding observation (14 months after ligation), as a small, fixed, hard, pulseless and silent mass in the infra-umbilical region, about size of a small mandarin orange. It was now evident that the clot in the sac had consolidated and the aneurysm had ceased to be active.

projected far above the iliac crest and reached the umbilical level. Roentgenograms showed marked erosion of the bodies of the third and fourth lumbar vertebrae, the promontory of the sacrum and the iliac crests. The diagnosis was then established of a *leaking aneurysm of the abdominal aorta at the bifurcation; involving both common iliacs;*

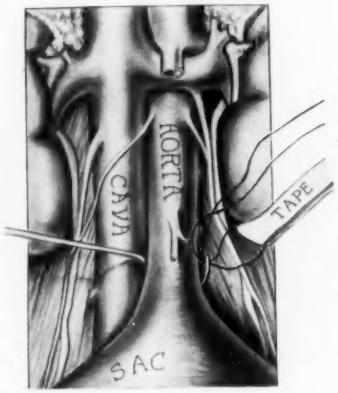

FIG. 7.—Ligation of the aorta immediately above the sac. One-half inch cotton tape led around artery by catgut traction loop and aneurysm needle.

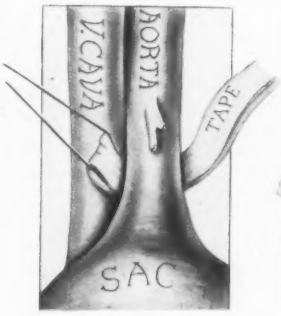

FIG. 8.—Continuation of Figure 7.

FIG. 9.—Tape cut in two and applied as double ligature to the artery. Upper tied first; shows first hitch in knot.

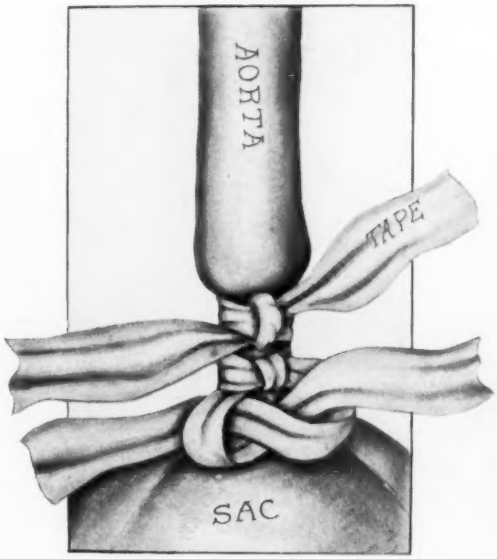

FIG. 10.—Both ligatures in place. Second knot being tied on lower ligature; second knot already tied in upper ligature.

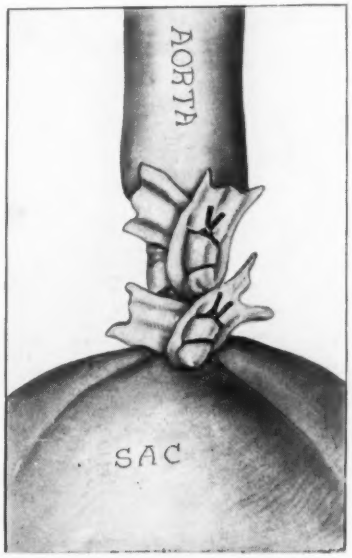

FIG. 11.—Both knots tied. Knots secured by through-and-through silk sutures to prevent slipping. The free edges of the tapes were cut short to reduce their bulk.

with progressive retroperitoneal extravasation. In view of the great suffering of the patient, and the evidence of a rapidly advancing retroperitoneal extravasation, the ligation of the abdominal aorta above the bifurcation was decided upon and by courtesy of Drs. Martin and King, the patient was transferred to Doctor Matas' service.

FIG. 12.—Closed sac filled with hard clot exposed in the open pelvis. The grooved director lies in the aorta as it enters the neck of the sac. 1, The right ureter displaced in front of the sac; 2, right iliac artery and underlying vein obliterated and lifted up by expanding sac; 3, left iliac vessels overlapped by extension of sac to left; 4, most prominent bulge of aneurysm at bifurcation, probable seat of original sac before leak began; 5, stiff wire spikes to indicate limits of extravasation when leak occurred.

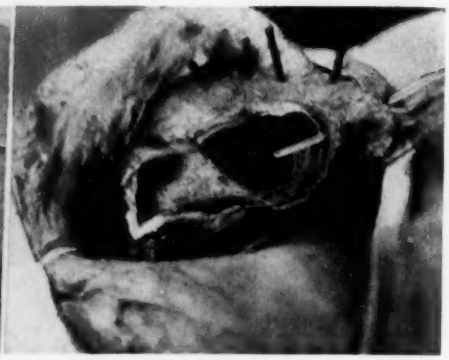

FIG. 13.—Aneurysmal sac shown empty of clot, filling the right iliac fossa, projecting over the brim of the pelvis, and extending over promontory of sacrum. Deep cavity at the bottom is a diverticulum of the sac projecting into and beyond the sacro-iliac notch. The stiff wire spikes indicate the outer boundary of the sac. The grooved director lies in the channel between the aorta and interior of the sac. The aorta is opened and stretched over a glass slide. A folded towel has been placed under the slide and neck of sac in order to bring the aorta into better view.

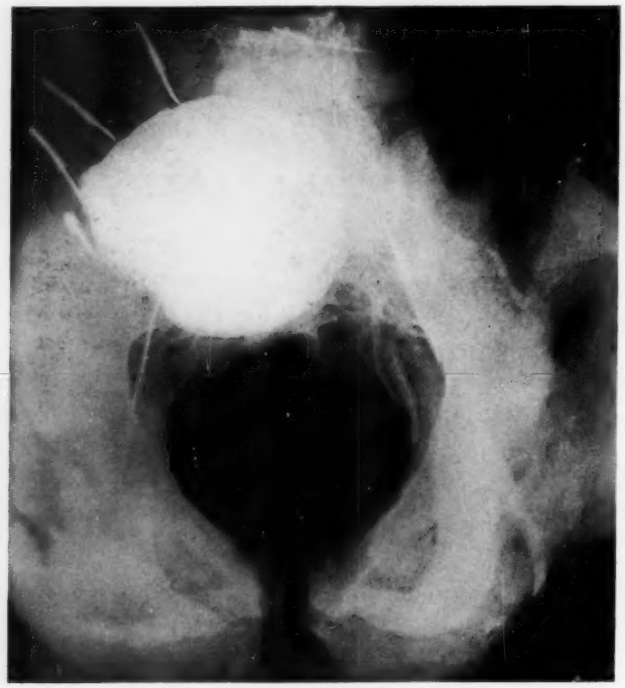

FIG. 14.—Radiograph shows the outlines of the sac clearly defined after it had been emptied of clot and the clot replaced by a radiopaque medium.

Operation.—April 9, 1923: Doctor Matas and Staff (Drs. L. H. Landry, S. Geismar, and A. Vidrine, resident intern, Miss Sawyer, chief nurse). Gas-oxygen anesthesia (Dr. E. E. Allgeyer.) Operation begun at 9:30 A.M. and finished at 10:30 A.M.

Synopsis.—Celiotomy in Trendelenburg position. Ligation of the abdominal aorta immediately above the sac with two *completely* occluding, one-half inch, cotton tape

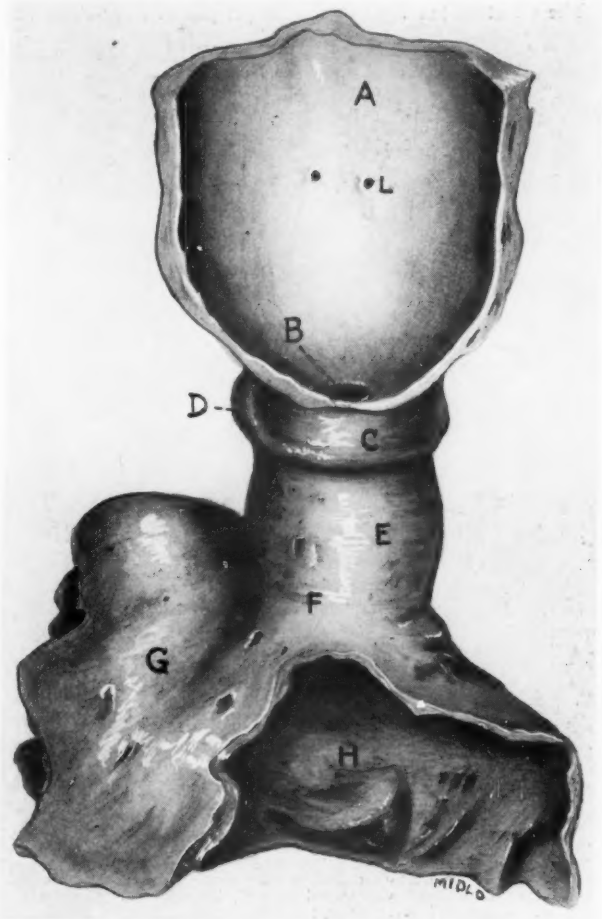

FIG. 15.—Semidiagrammatic sketch of lower abdominal aorta at the ligation; anterior view. A, Aorta cut open as far down as ligature; B, newly formed lumen; C, position of cotton tape ligatures; D, bulge due to knots; E, pouchlike portion of aorta betwen ligatures and internal tissue shelf at F; G, surface, and H, interior of aneurysmal sac which has largely been cut away; L, orifice of fourth lumbar artery.

ligatures (sterilized and fat free by preservation in ether). The tapes were placed in juxtaposition, one above the other. Upper tape tied first, with first knot secured by silk sutures passed through the knot to prevent slipping. Before tying the second knot the effect of the occlusion upon the aneurysm was observed, and it was found that the sac had collapsed and all pulsation had ceased. The first knot was tightened with just sufficient force to obtain a complete occlusion without crushing the walls of the artery. The second (lower) tape was then tied and the knots secured by sutures in the same way. A circular

area of the artery, a little less than one inch in breadth, was thus embraced by the two ligatures. The parietal peritoneum was then sutured over the aorta and the abdomen closed. The appendix, which was long, thick, and beaded with fecal calculi, hung over the aneurysmal sac, and was excised.

Immediate Operative Result.—The immediate effect of the ligation was to increase the pulse rate from 121 to 154, and the blood pressure was lowered from 115/85 to 100/70. As the abdomen was closed, the pulse was 160; blood pressure 100/70, respirations rose from 24 to 38. The effect of the ligation on the peripheral circulation was to suppress

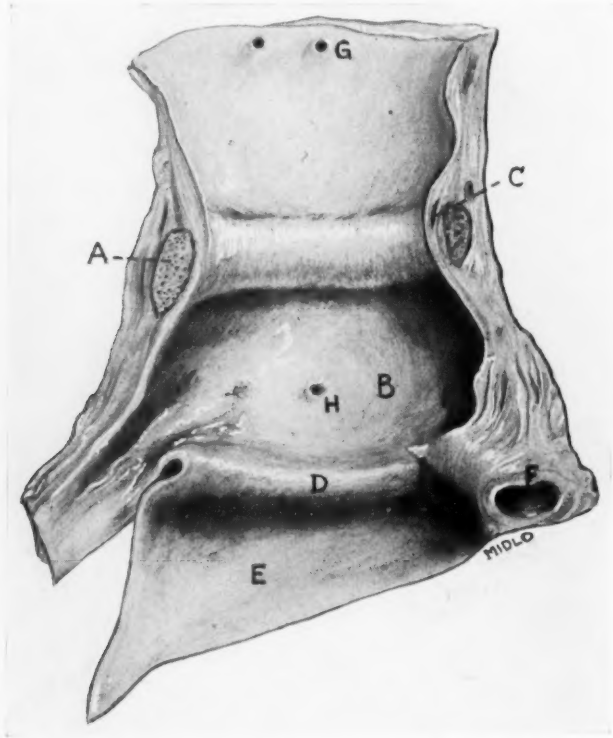

FIG. 16.—Internal view of lower abdominal aorta at the ligation. The anterior wall of the aorta and sac have been cut longitudinally to expose the lumen surface. A, cotton tape ligatures; B, pouch-like portion of aorta between ligature and internal tissue shelf, D; C, area of calcification; E, wall of aneurysmal sac; F, left common iliac artery; G, orifice of fourth lumbar artery; H, orifice of middle sacral artery.

the femoral and pedal pulses, but the feet retained their sensibility, warmth and living color. Four days after the operation the respirations rose from 40 to 50, coincidently with the clinical signs of a lobular bronchopneumonia, which appeared in edematous patches in both lungs, accompanied by a profuse mucosanguineous expectoration. The patient continued in a precarious condition, with threatened cardiorespiratory failure, until the ninth day, when relief came with the return of pulsations in the aneurysmal sac and in the femoral arteries in the groin.

Postoperative Course.—The postoperative history is one of early relief from pain and rapid reduction in size and activity of the aneurysm, gradual, general improvement in weight and strength under the influence of rest, improved nutrition and specific medication until about April 20, 1924, when signs and symptoms of *progressive tuberculous infection* began to assert themselves in the cervical lymph nodes and in the lungs. Chains

of caseous tuberculous nodes were twice extirpated, but a rapid infiltration of the lungs with breakdown into cavities, profuse mucopurulent expectoration laden with tubercle bacilli, and "hectic fever" completed the picture of a "galloping phthisis florida," which culminated in a fulminating pulmonary hemorrhage, and death on September 10, 1924. During the year, five months and nine days that the patient survived the ligation of the abdominal aorta, the reduced circulation in the sac, enforced rest, and the extremely low blood pressure that characterized the postoperative period, all combined to promote the deposition of laminated clot and progressive consolidation of the sac.

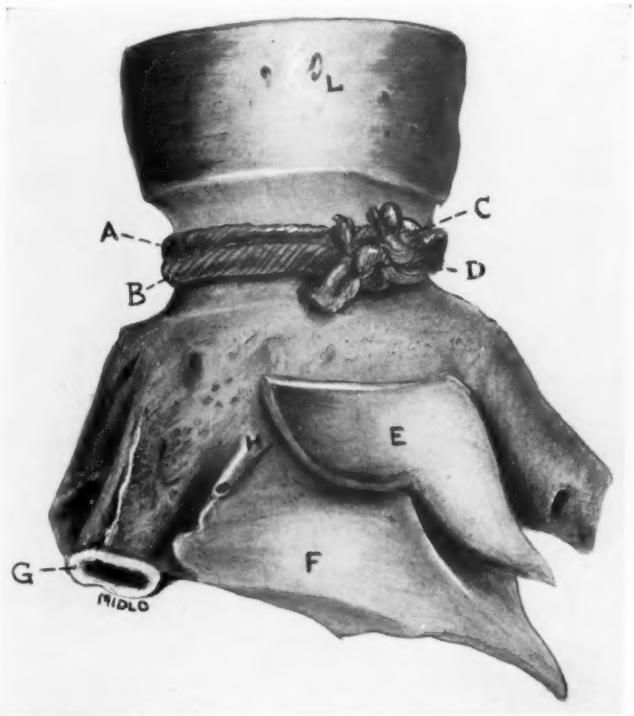

FIG. 17.—Posterior view of lower abdominal aorta at the ligation. The anterior wall of the aorta and sac have been cut longitudinally. The entire outer aspect of the vessel wall is shown. The tissues covering the outer surface of the ligatures have been dissected away, exposing the two pieces of cotton tape A and B, and their knots, C and D; E, a small portion of the inferior vena cava; F, the upper portion of the aneurysmal sac; G, the left common iliac artery; H, middle sacral artery; L, orifice of fourth lumbar artery.

On April 11, 1924, about one year after the ligation, the aneurysm had contracted fully 60 per cent of its original size, and pulsations could be felt only in small, restricted areas. Three months before the patient's death, the aneurysm had solidified and had lost all of its aneurysmal characters; it had become an inert and symptomless pelvic tumor. The patient died with her aneurysm clinically cured.

REMARKS.—Immediately after death every precaution was taken to preserve the body and prepare it for a most searching roentgenologic and anatomic study. The roentgenologic and photographic studies were undertaken at the roentgenologic laboratory of the Charity Hospital, with the valued cooperation of the late Chief, Dr. A. Granger. The histology and photomicrography

FIG. 18.

FIG. 19.

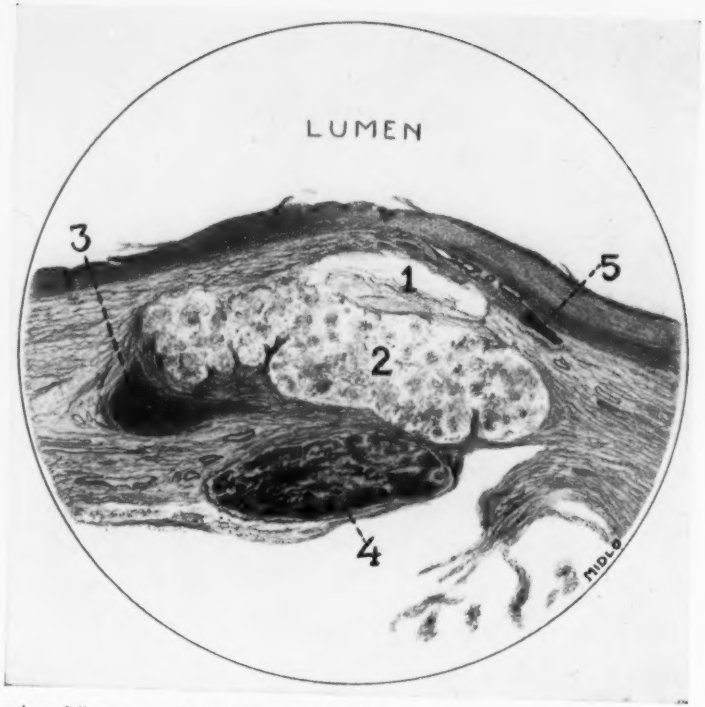

FIG. 18.—Photomicrograph of a longitudinal section of abdominal aorta in the region of ligatures. (×25) Hematoxylin-eosin.
FIG. 19.—Drawn from photomicrograph (Fig. 18). 1, Necrotic zone probably produced by pressure interference of blood supply; 2, cotton tape ligatures surrounded by connective tissue and infiltrated with fibrous connective tissue and foreign body giant cells; 3, denser zone of connective tissue, probably repair process in area of needle passage; 4, small hemolymph node; 5, area of calcification. (×25) Hematoxylin-eosin.

FIG. 20.

FIG. 21.

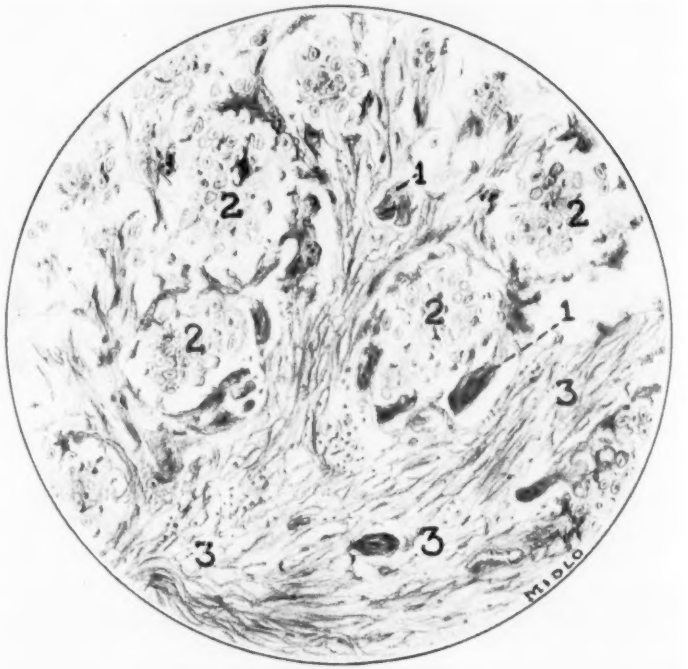

FIG. 20.—Photomicrographs of an area of the longitudinal section of the abdominal aorta in the region of ligatures showing some bundles of cotton fibers surrounded by and infiltrated with foreign body giant cells; also, an area of new-formed connective tissue, resulting from organization of exudate. (×250)

FIG. 21.—Drawn from photomicrograph (Fig. 20) showing several strands of the cotton tape surrounded by and infiltrated with fibrous connective tissue and foreign body giant cells. 1, Giant cells; 2, strands of cotton tape, sectioned transversely; 3, connective tissue and giant cells in organized exudate. (×250)

FIG. 22.

FIG. 23.

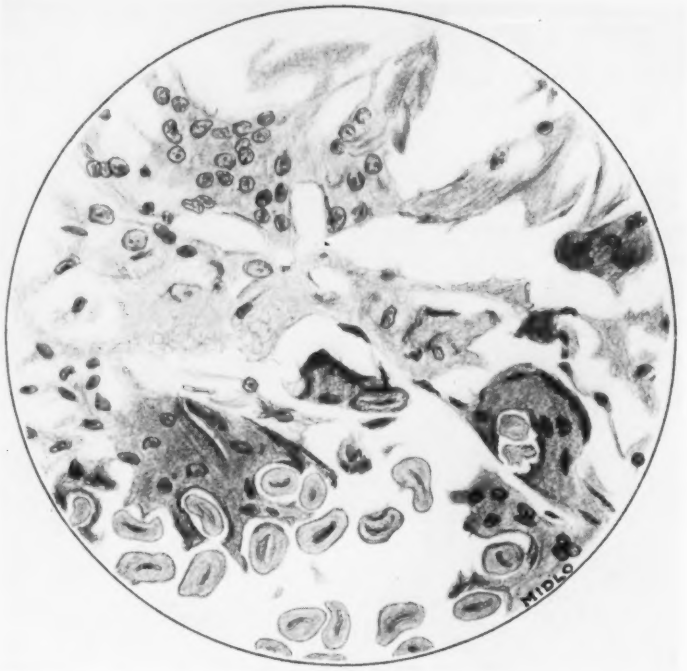

FIG. 22.—Photomicrograph of a small area of the longitudinal section of abdominal aorta in the region of ligatures showing some fibers of the cotton tape, and large foreign body giant cells, some of which have engulfed cotton fibers. (×500)

FIG. 23.—Drawn from photomicrograph (Fig. 22) showing a number of individual cotton fibers belonging to a strand of the tape used in the ligation; and foreign body giant cells, some of which show engulfed cotton fibers. (×500)

were recorded in the laboratory of Professor I. Hardesty, of Tulane University by Dr. Charles Midlo, his able and painstaking assistant (now assistant Professor of Anatomy, Louisiana State University), to whom I am especially indebted for all the photomicrographs and histologic drawings exhibited in this series (Figs. 15 through 22).

The details of the procedure adopted for embalming and injecting the arterial system with radiopaque fluid (a thin colloidal bismuth emulsion) have been described in the author's previous publications, and are the first in which roentgenology has been utilized to visualize (postmortem) the arterial system after a ligation of the abdominal aorta for an aneurysm of this artery. This is, also, probably the first case in which the changes in the ligature and in the tissues of the aorta at the site of ligature have been microscopically studied and photographically recorded.

Among the more salient conclusions that may be drawn from the clinical and postmortem studies of the case are:

(1) The patient died 17 months and nine days after the ligation of the abdominal aorta for a leaking (ruptured) syphilitic aneurysm of the abdominal aorta at the bifurcation, including both common iliac arteries.

(2) The cause of death was tuberculosis—a cause unrelated to the aneurysm.

(3) That the collateral circulation *above and below the aneurysm* was well established before the ligation of the aorta.

(4) That the patient had been clinically cured of the aneurysm, and that this had ceased to be an active factor in her invalidism fully three months before her death.

(5) The clinical evidence of cure was fully confirmed at the postmortem by the complete consolidation, contraction of sac contents, and beginning organization of the clot.

(6) The invalidism and general disabilities, that hospitalized the patient until her death, were caused by the ravages of a disseminated wide-spread pulmonary, lymphatic and joint tuberculosis, which flourished with unusual rapidity and luxuriance in a soil seemingly fertilized by a saturating and malignant luetic infection.

(7) The aorta was totally occluded for nine days following the ligature, during which all pulsation ceased and the peripheral pulses in the femoral and pedal arteries were suppressed.

(8) During this period of total occlusion, the patient remained in a critical condition from threatened cardiac and pulmonary failure (passive congestion, patchy lobular pneumonia, pulmonary edema), which was only relieved by the yielding of the ligatures sufficiently to allow a small, reduced stream to flow through the ligated segment, thus converting a total *atresia* into a partial, *stenotic* occlusion.

(9) The yielding or relaxation of the ligatures was not caused by any slipping of the knots but, as demonstrated at autopsy, by the soaking of the

cotton fibers in the tissue juices, and the permeation and erosion of the fibers by giant foreign body cells.

(10) The reduction of the aortic stream to about one-tenth or one-eighth of the caliber of the normal aorta was conducive to the final cure of the aneurysm by favoring a gradual deposition of clot and consolidation of the aneurysmal sac.

(11) The anatomic and histologic studies of the aorta at the seat of the ligature showed, conclusively, that the cotton tape ligatures employed in this case (tightened without crushing force) were well tolerated by the tissues and caused no damage to the artery.

(12) As shown in Figures 7–11, the two one-half inch cotton tapes remained imbedded and incorporated in the aortic walls as a constricting ring for over 17 months without causing the slightest ulcerative, necrotic or thrombotic changes in the arterial coats and especially the intima which remained well-lined and polished with normal endothelium.

(13) This experience shows that a partial occlusion can cure an aneurysm of the terminal aorta slowly, but with greater safety than an immediately total occlusion, without cutting through the artery or causing ulcerative alterations in the intima that might lead to hemorrhage or thrombosis.

(14) It would seem that in large and leaking aortic aneurysms, with progressive subperitoneal extravasation, the collateral circulation is well-established. In such cases the immediate total occlusion, which is especially indicated to stop the leaking, may probably be better tolerated than in the earlier and nonleaking aneurysms, in which the collateral circulation has not had time to develop.

(15) In view of the fact that sterile cotton tape is so well tolerated by the tissues and is ultimately incorporated by the aorta in the structure of its walls, it would seem unnecessary, and superfluous, to resort to extemporized autogenous fascial strips or to heterogeneous aponeurotic or other membranous strips, kept in stock, when the cotton tape will answer the same purpose with greater simplicity and safety.

(16) Judging by the recent experimental evidence and the increasing number of clinical cures of aortic aneurysms by ligation and by suture methods, and the interesting evidence recently furnished by the laboratory, it would seem reasonable to expect that the great desideratum of abdominal aortic surgery, namely, the safe occlusion of the aorta *in any part* of its abdominal and low thoracic course, by gradual methods of occlusion (Owings) will ultimately become as feasible and legitimate in the surgical clinic as in the experimental laboratory.

EXPERIMENTAL STUDIES ON THE GRADUAL OCCLUSION OF LARGE ARTERIES*

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THERE is no completely satisfactory method for the gradual occlusion of the great vessels despite repeated attempts to perfect one, for, though many ingenious instruments have been devised, all of them have some drawback. Such a method is desirable in order to treat aneurysm and other lesions of large arteries.

All of the early devices to shut off large arteries used some sort of external compression on the wall. The first of these was with a ligature, but when it was realized that gradual occlusion was necessary, clamps were used that pressed on the vessel indirectly from the outside, often impinging it against a bone. Haberland[8] shows some of these devices and their method of application. Next, tapes, tubes, clamps or snares were used that encircled the vessel, protruded from the wound and were tightened from the outside. According to Matas,[12] the concept of gradual arterial obliteration was originated late in the eighteenth century. Deschamps[5] was one of the first to believe "that by gradual obliteration the collateral circulation would be developed sufficiently to diminish the danger of gangrene." He encircled the artery with a wide tape which was held against a flat metallic plate, against which the artery was compressed by tightening the tape with a snare. Dubois[7] was the first to put this principle into practice by compressing the parent artery of an aneurysm. Assolini[1] devised a spring forceps in which the compression of the artery was regulated with a screw. Subsequently, a great many instruments for arterial compression were made, among which were those of Cooper,[3] Doberauer,[6] Jordan,[10] Keen[11] (Fig. 1), Riese,[22] Smoler,[23] and Stratton.[24] It was proposed by Milton[15] that a rubber tube be placed to encircle the vessel and be tightened from the outside (Fig. 2). All of these devices had two major disadvantages: that of infection traveling along the sinus from the outside down to the vessel, and that of hemorrhage both from compression atrophy of the vessel wall and from secondary infection about the artery.

Halsted[9] was the first to overcome the hazard of infection by the introduction of his aluminum band (Fig. 3). This was one of the most important contributions to vascular surgery, for it established the concept of aseptic gradual occlusion. In regard to it he says: "The notion of gradual compression in the ordinary use of the term was entertained, only to be definitely discarded, because of the seemingly insurmountable difficulty of preserving asepsis. A sinus must form about any instrument leading from the aorta to

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the air and, sooner or later, such a sinus necessarily becomes infected." Infection predisposes to secondary hemorrhage.

Soon after this, Matas[13] proposed the use of a heavier aluminum band to be clipped onto arteries to compress them, and thus test the efficiency of the collateral circulation (Fig. 3). It was soon used as a means of partial occlusion preliminary to complete obstruction of large arteries. Because this band is malleable, it can be applied without a special instrument so is often used to-day instead of the Halsted band.

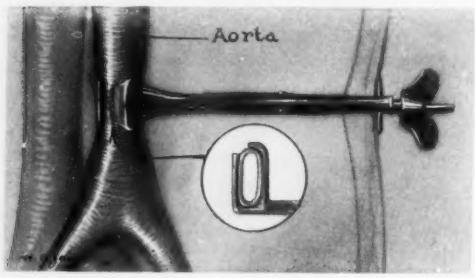

FIG. 1.—The Keen[11] clamp for gradual arterial closure was one of many of the earlier devices employed for this purpose.

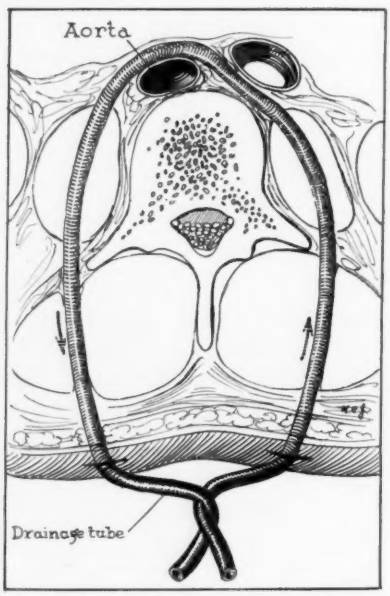

FIG. 2.—An early method of gradual occlusion of the aorta was suggested by Milton,[15] using a rubber tube passing over the vessel and out through incisions on either side of the spine.

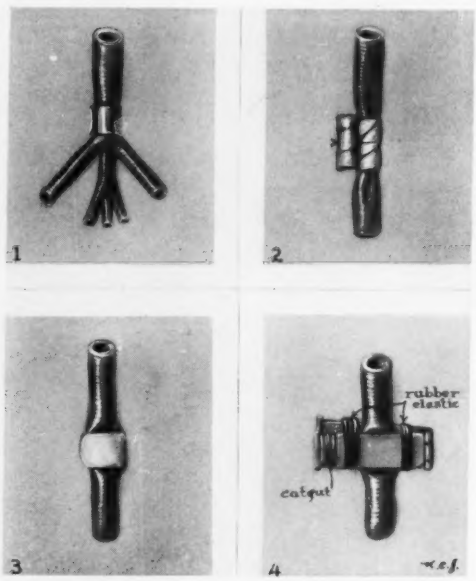

FIG. 3.—Methods of aseptic, gradual arterial occlusion by external compression include: (1) The Halsted[9] band. (2) Fascial strips. (3) The Matas[13] band. (4) The Neff[16] clamp. These all have the disadvantage of causing pressure atrophy and possible rupture of the vessel.

Neff[16] devised an ingenious clamp for gradual, progressive arterial obstruction (Fig. 3). He hinged two metallic strips at one end, placing the vessel between them. The opposite ends were held apart by strands of plain catgut or decalcified bone, while at the same time elastic bands tended to approximate them. As the catgut dissolved, the force of the elastic closed the two metallic strips, compressing and obstructing the artery between them. In the experiments reported by Neff in which this clamp was applied to the dog's aorta, hemorrhage or infection occurred in all instances.

Finally, ligatures, tapes and tissue bands have been used unsuccessfully to partially obstruct the large arteries. Halsted comments on his experiments as follows: "Fine silk cut through in two days; coarse silk cut through more slowly, knotted ligatures were found to be unsuitable, for a desired degree of constriction or obliteration could not be accurately obtained nor could the crushing of the arterial wall be invariably avoided. Tapes of various materials were tested—of cotton, of chromicized intestinal submucosa, of elastic tissue obtained from the aorta, of aponeurotic white fibrous tissue. These tissue bands always relaxed and allowed reestablishment of the lumen." This has been the finding of others[18] who have tried them.

Experience has shown that all methods that compress the vessel will eventually cause it to atrophy. The expansile pulsation pounding against the compressing device weakens the wall and may even cause rupture with fatal hemorrhage. Halsted recognized this danger, for he said: "The experimental work on animals had led me to expect that ultimately the metal band must cut through the artery, because in cases observed seven months or less, the wall of the aorta had become atrophied to the thinness of paper and there was no adhesion between the infolded attenuated surfaces." The histologic studies of Reid[20] showed this atrophy to be due to loss of elastic and muscular tissues. Later, Reid[21] made the statement that "partial or complete occlusion of a large artery by compression always leads to the death of the vessel wall."

It appears incontrovertible that externally compressing devices are dangerous. A consideration of the other alternatives leads to the conception that vessels might be obstructed by internal thrombosis, intrinsic contracture of the wall or extrinsic contracture of scar about the wall. These principles have been tested in the following experiments:

EXPERIMENTAL STUDIES

All experiments in this field must be interpreted in the light of known differences between the effect of arterial obstruction in animals and in man. The dog shows little or no ill effect from abrupt ligation of one or both carotid arteries, while in the human this is followed by cerebral complications in one-third of the cases. The same difference in response holds for the ligation of most other arteries. Occlusion of the aorta in animals is more comparable to that in the human. This is especially true of the thoracic portion, for here sudden ligation invariably causes death in a few hours, the wall is weak and prone to rupture, and the development of collateral circu-

lation is vital. For these reasons the thoracic aorta of animals is the proving ground of methods for use in man. It is assumed in this discussion that a method that is effective on the aorta will be applicable elsewhere.

Methods of Internal Obstruction.—Intravascular occlusion was first tried by Reid[21] with the use of fascial plugs, in order to avoid the atrophy from external compression. He says: "For such a method of occlusion there would seem to be no practical demand, for the arteries that commonly need to be occluded in the human being can be occluded by proper ligation. However, some modification of the method, as for example the introduction of a fascial ball with a small tube through its center, may be the best way of producing a partial occlusion of the human aorta." Carrel[2] and others had placed tubes in the vessels only to find that they usually plugged with a thrombus; but this result could be put to use if the rate of thrombotic occlusion was slow enough to permit collateral channels to dilate. Several years ago such a method was proposed[19] which was based upon this principle. In order to avoid the hazard of opening the artery to insert a tube, a coiled tubular spring was introduced through a puncture wound and screwed into the lumen. This caused a gradual closure by thrombosis. In the specimens examined several months after operation, a small canal had opened through the thrombus but a major degree of obstruction persisted. This condition is apparently permanent, for it was demonstrated on one dog, observed for seven years after operation, that only this small canal was present (Fig. 4). This animal was active and healthy, being able to run about without apparent fatigue, although the femoral pulses were absent. It was interesting to note, also, that the animal would shiver only in the hind legs in cold weather. These were the only abnormal signs, yet when the specimen was examined it was found that the greater part of the circulation to the lower half of the body was passing through collateral channels.

FIG. 4.—The specimen removed from a dog who had a spring inserted into the thoracic aorta seven years previously. A major degree of occlusion has persisted, since only a small orifice reformed through the original thrombus. The spring steel was intact and showed little evidence of corrosion.

This method of intravascular obstruction has the disadvantage of technical difficulty in inserting the spring when the vessel is in a deep wound. The device has to be turned parallel to the axis of the artery before it can be completely screwed into place and this manueuver is sometimes hard to accomplish without causing bleeding. To overcome this, a flat spring, such as is used in the mainspring of a clock, was tried. It would curl up in the lumen

to form concentric rings and create a grid across the orifice. This did not work because too much obstruction was caused by the metal and too rapid thrombosis resulted. The flat type of spring produced linear barriers across the lumen rather than forming a tube-shaped structure within it.

Another device tested for this purpose is shown in Figure 5. It consists of a metal cuff which contains 12 sharp projections on its inner wall. This was suggested by Dr. Beverly Raney, who thought that if it was placed snugly

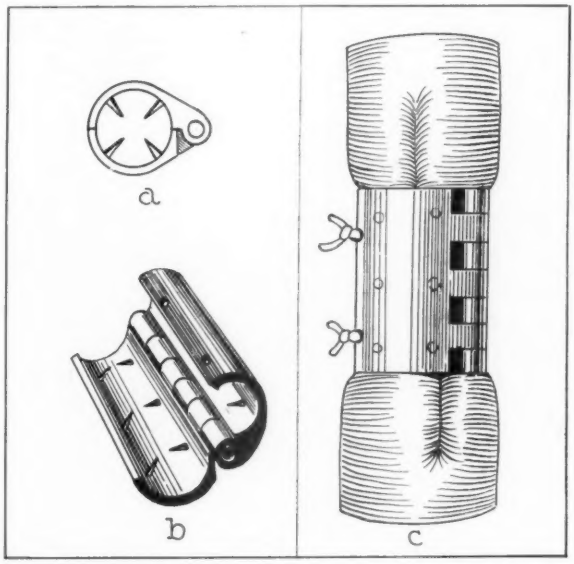

FIG. 5.—This device contains 12 prongs which were designed to slowly perforate the artery when the clamp was closed over the vessel. The prongs did penetrate the arterial wall but did not do enough damage to cause occlusion by thrombosis.

around the artery the prongs would gradually be forced through the wall and protrude into the lumen, causing thrombus formation. This was a good idea but it did not work, for the penetration of the prongs through the vessel wall was too slow to cause the necessary damage.

Injury to the intima by sclerosing solutions did not cause thrombosis. It was not expected that they would, but they were tried just to make sure that such irritating solutions as sodium morrhuate or 50 per cent glucose would have no effect. A segment of the thoracic aorta of dogs was isolated with rubber-shod clamps, filled with the sclerosing solution, and after one to five minutes the circulation was restored. No obstruction resulted, for it requires not only damage to the intima but also slowing of the stream to produce thrombotic occlusion of a large artery.

Intrinsic Contracture of the Vessel Wall.—The first attempt to reduce the caliber of the aorta by constriction of its wall was made by Matas and Allen,[14] using plication sutures. They reported experiments in 151 dogs in which mattress sutures were used to narrow the vessel. This plication was increased

at a second- and third-stage operation until, in some instances, the lumen was nearly obliterated. Reid[21] performed similar experiments and studied the effect of the sutures on the arterial wall. It was found that relaxation gradually occurred and some restoration of the size of the lumen resulted. In some human cases attempts to suture the diseased wall of the aorta have resulted in hemorrhage.

It is conceivable that sufficient damage to the wall of an artery would result in its eventual contracture with closure of the lumen. This hypothesis was tested by three methods: Injection of irritants into the wall; painting caustics on the outer surface of the wall; and coagulation of the vessel with a diathermy current.

Sclerosing solutions, principally sodium morrhuate, were injected into the wall of the thoracic aorta of dogs, using a very small needle inserted beneath the adventitia. Injection with force causes a ring of solution to infiltrate around the circumference of the artery. The specimens were studied at intervals of from two weeks to three months after injection, and no narrowing of the caliber of the vessel found. In one instance a typical arteriosclerotic plaque occurred on one side of aorta, apparently resulting from the necrosis of the wall at the time of injection.

In another series of experiments, the thoracic aorta of dogs was exposed and a segment denuded of all surrounding tissue (Fig. 6). This part was painted with full strength iodine solution, 35 per cent silver nitrate or 25 per cent aqueous acriflavine. No change occurred in the caliber of the vessel either soon or some time after this treatment. The specimens showed some scarring about the aorta with adhesion of the pleura or lung to the operative site, but the wall of the artery was intact and supple.

The best method of injury to the arterial wall appeared to be with a diathermy current. Twenty experiments were undertaken upon the aorta and carotid arteries by electrocoagulation. The vessel was exposed, denuded, and lifted up by a band of lead foil 3 Mm. wide (Fig. 6). A coagulating current was passed through this which traversed the two sides of the loop of exposed vessel, gradually coagulating it to a white color. Overcoagulation turns the tissue black and causes it to shrivel to a cord. This can be prevented by using a low milliamperage, allowing some blood to pass through the loop to cool it during coagulation and by careful application of the current. Two machines of Liebel-Flassheim make were used: the "Bovie" and the "Electric Scalpel" models. They were set at low voltage, medium dehydration, and power control of 25 to 30.

The results of these experiments on electrocoagulation of the arterial wall were disappointing. At the time of operation the vessel was constricted to about one-third of its normal size in two places on either side of the contact of the positive electrode. There was no pulse distal to this point but a well-marked thrill was present. The specimens were then examined at intervals of from two to eight weeks after operation. In three experiments overcoagulation occurred with complete occlusion of the lumen by thrombosis.

This apparently resulted soon after operation. In all others there was a gradual, progressive return of the caliber of the vessel to its normal state. This was usually complete in about a month, after which little or no evidence of injury persisted.

It is seen that in all of the experiments where gradual occlusion was attempted by damage to the vessel wall, the injury was insufficient to cause

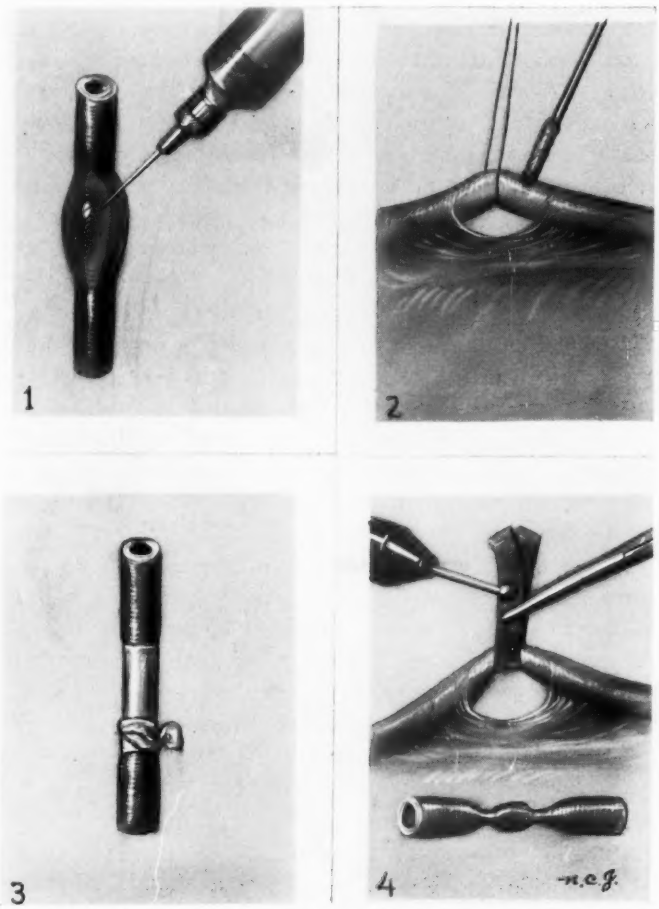

FIG. 6.—Methods of injury to the arterial wall in an attempt to induce spontaneous closure from contracture included: (1) Injection of irritants into the wall. (2) Painting irritants onto the wall. (3) Wrapping cellophane about the vessel. (4) Slow coagulation of the wall with diathermy.

permanent constriction of the artery. In fact, that produced at operation eventually relaxed and allowed restoration of the normal caliber. In view of this, it is probable that the reparative capacity of the artery is great enough to correct a sublethal damage, so the attempt to produce gradual progressive occlusion by this means is unsatisfactory.

Occlusion by Extravascular Contracture.—Theoretically, an artery could be shut off by the contracture of a large amount of scar tissue about its wall.

The first attempt to do this was with strips of fascia[18] which were wrapped around the aorta, making a concentric ring of fibrous tissue about it. This fascia gradually relaxed and eventually absorbed so that no evidence of obstruction existed after two or three months.

Cutler[4] has used acriflavine to cause fibrotic occlusion of the bronchus. This substance causes fibroplasia in tissue, so might be used to constrict arteries with scar tissue. Acriflavine, in a 25 per cent aqueous solution freshly prepared before each experiment, was injected in the tissues about the thoracic aorta of dogs. It was infiltrated around the vessel for a distance of about one inch along its course. The specimens were examined at intervals of one, two and three months and no contracture found that was sufficient to constrict the aorta. Only six experiments were undertaken, so it may be worth while testing this compound more thoroughly. If greater periarterial infiltration could be accomplished or repeated applications made, it might produce more effect.

Silica is known to produce extensive scarring in pulmonary tissue causing pneumoconiosis. How much fibroplasia it causes in other tissues is not so well known. Recently, it was decided to try placing silica, in finely divided particles, into the tissues about the aorta in an effort to cause extensive scarring around the vessel. There are insufficient data available at present to judge the results of these experiments, which are being continued.

Page[17] found that cellophane wrapped loosely about the kidney caused an excessive reaction in the tissues, constricting the kidney and causing hypertension in dogs. It is conceivable that if the reaction from cellophone was great enough to contract a kidney it would be sufficient to squeeze down the caliber of a large artery.

Twenty-four experiments were undertaken by wrapping cellophane about the aorta or carotids of the dog. Ordinary DuPont cellophane No. 300 P.T. was used that was soaked in alcohol or mercury oxycyanide solution at least for 12 hours prior to operation. It was folded into a strip four layers thick and loosely wrapped about the vessel three times. The ends were tied to hold them in place or a silk ligature was placed around the ends. Every effort was made to avoid external compression on the artery but in spite of this the cellophane was tight enough to cause rupture of the aorta in one animal.* Another dog died of a pulmonary infection soon after operation. The remainder were satisfactory for evaluation of the method.

Cellophane was found to be an extreme tissue irritant, for, with but two exceptions, an extensive change occurred around it. This consisted of an intense reaction with either purulent or gelatinous fluid in the center about which the tissue contained many phagocytic, mononuclear cells interspersed among fibrous tissue. This fibrocollagenous layer became partly hyalinized. This process causes a steady progressive constriction of the vessel and an eventual obliteration of the lumen in some instances. Figure 7 shows, by means of latex injection casts, how the caliber of the abdominal aorta was

* In a subsequent series of experiments two other dogs have died of rupture of the thoracic aorta.

diminished by the tissue reaction from cellophane wrapped about it. As the process continues there comes a time when the intima is involved and destroyed, shutting off the vessel completely (Fig. 8).

This is the first time that gradual obstruction of the aorta has been deliberately produced by perivascular irritation. The only drawback to the method is the intensity of the reaction caused by the cellophane. It makes one hesitate to use it in patients, yet apparently a very extensive irritation is necessary to shut off these large arteries since lesser degrees of damage have no effect. Perhaps the amount of cellophane applied could be reduced to a point where it was just sufficient to cause occlusion but not enough to create such an extensive reaction in the surrounding tissues. This is being tried.

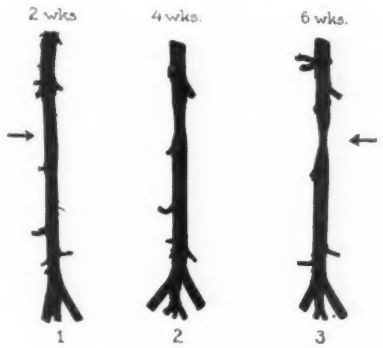

FIG. 7.—Gradual, progressive constriction of the dog's abdominal aorta by contracture from cellophane irritation is demonstrated by these latex injection casts of the vessel. The arrows point to the level of injury.

The importance of these observations lies in the demonstration that gradual occlusion of the dog's aorta can be produced by a fibroplastic reaction in and about the vessel wall, thus illustrating the feasibility of such a method.

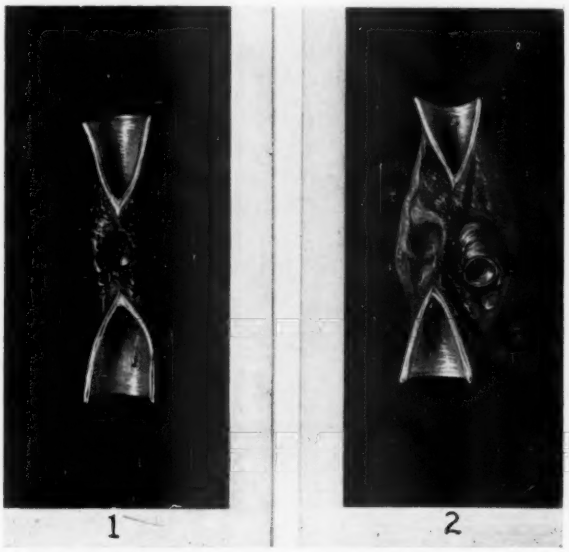

FIG. 8.—Two specimens of complete occlusion of the dog's aorta by cellophane wrapped loosely about the vessel. The first one has a central crater which contained fragments of cellophane, while in the second the cellophane retained its circular form but migrated to one side of the artery.

SUMMARY.—During the last century many attempts were made to produce gradual progressive occlusion of the aorta and other large arteries by means

of bands, clamps or ligatures which compressed the vessel. All experience has shown that the force of the pulse pounding against the compressing device will weaken and rupture the arterial wall. All methods that depend upon external compression are dangerous.

Theoretically, there are at least three other methods by which large arteries can be gradually obstructed: By internal occlusion with thrombosis; by intrinsic contracture of the vessel wall after its injury; and by extrinsic contracture of scar tissues around the artery. These have been tested experimentally.

About 12 years ago, a method was reported for closing the thoracic aorta of dogs by thrombosis caused by a spring screwed into its lumen. Continued observation of these animals, one of them for seven years, reveals the persistence of a major degree of obstruction. The particular virtue of the spring is the tenacity with which it holds the thrombus by the multiple coils within the lumen. This not only prevents embolism but also gives a more permanent occlusion. The disadvantage of the spring is the difficulty of inserting it into the vessel in a deep wound, but, thus far, no better method of intravascular closure has been found.

Intrinsic contracture was attempted by injury from iodine, silver nitrate, sodium morrhuate or acriflavine painted on the arterial wall or injected into it. This caused no narrowing of the vessel. Diathermy coagulation resulted in temporary constriction but eventual restoration of the lumen to a normal caliber. There is apparently too little damage produced by these means to cause occlusion or even permanent constriction of the aorta.

Extrinsic contracture of the perivascular structures was first attempted by the use of large amounts of fascia but this relaxed rather than contracted. Acriflavine and silica have been tried without effect as yet.

A striking result followed the use of cellophane wrapped loosely about large arteries. An intense perivascular irritation was produced by the cellophane, with first constriction of the vessel and then invasion and destruction of the intima resulting in complete closure. This occurred even with the aorta. It is the first time that complete occlusion has been produced by the extravascular contracture.

CONCLUSION

Closure of the aorta both by intravascular thrombosis and by extravascular irritation has been demonstrated. It remains to perfect the methods by which this is done, for they can, in all probability, be improved.

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DISCUSSION.—DR. EMILE HOLMAN (San Francisco, Calif.): We are, indeed, grateful to Doctor Pearse for giving us another method of treating large arteries when we are confronted with the necessity of occluding them.

I am reminded of Van Allen's suggestion that patients might be prepared for lobectomy by wrapping the affected lung in cellophane, waiting for fibrous contraction to occur, and then, subsequently, performing a lobectomy. This was done in experimental animals with great success, indicating that there is a very definite stimulating effect of cellophane upon the production of fibrous tissue.

I can visualize one site where such an irritating substance might be used to advantage. I recently operated upon a young child with a patent ductus arteriosus. There was definite improvement, as shown by a rise in diastolic pressure and a decrease in dyspnea. However, a thrill recurred two days after operation which persists to the present time, and I am convinced that there has been partial restoration of the patent ductus. Is it not possible that, at this site, one could use some such gradual occluding agent?

With reference to the ligation of large arteries in continuity, attempt has been made by operators to prevent the erosion of the artery by using larger and larger occluding ligatures. However, they all have the inherent danger of the fact that this ligature is applied at a fixed point and that the distending force of pulsation is directed at this point with each beat of the heart, with gradual rupture of the tissue and rupture of the vessel. When we divide a large artery, as, for example, the abdominal aorta in the dog, there is a retraction of 3.8 cm., and separation of the divided ends due to elasticity.

Following division of the artery, there is no fixed point. The force of each pulsation is used up in the lateral expansion and in the lengthening of this proximal segment, so that there is no tendency for rupture of the tissues at the point of ligation. I think we should endeavor, in every instance, to divide a large artery between ligatures rather than to ligate it in continuity.

DR. FREDERICK L. REICHERT (San Francisco, Calif.): I simply wanted to ask Doctor Pearse if the work of Doctor Van Allen, published some five or six years ago, in which he demonstrated the use of rubber, silk, and, I think, cellophane, around large arteries, was not much the same as he has done.

DR. RUDOLPH MATAS (New Orleans, La.): In dealing with the gradual occlusion of the aorta and its primary branches, Doctor Pearse has brought us one of the most important problems in vascular surgery. He has reviewed all the important evidence on this subject very graphically and instructively. Unfortunately, and despite the great study, scientific ingenuity and experimental research that has been given to this problem, a safe, practical method of gradual occlusion of the human aorta has remained an unfulfilled desideratum.

In 1910, a great wave of experimental activity in the surgery of the vascular system had spread all over the world in consequence of the new impetus given to blood vessel technics by Carrel's improved method of suture. At the same time, the increasing incidence of aortic aneurysms as they were being disclosed by roentgenologic examinations intensified the search for methods of cure and relief by an improved surgery. The chief objectives of the experimental laboratories were the thoracic and abdominal aortas, in which the failure of the immediate, total ligature had led to a better prospect by the methods of gradual occlusion.

It was in this way that, in 1910, and contemporaneously with many other investigators (Haecker, Carrel, Halsted, Guleke, Jager, Lawen and Sievers, Schuppelmann, *et al.*), I undertook a long series of experiments in association with my friend and most valued assistant, the late Dr. Carrol W. Allen*

* Matas and Allen: Trans. Am. Surg. Assn., **31**, 195–217, 1913. *Idem:* ANNALS OF SURGERY, **58**, 304–319, September, 1913.

in which we aimed at the gradual occlusion of the thoracic aorta (below the left subclavian) by a series of plications, or infoldings, of the aortic walls which were turned into the lumen of the aorta, thereby reducing its lumen in proportion. The plan was to undertake the infolding at intervals—in about three stages. In the last stage, the infoldings filled the artery completely and transformed it into a solid cylinder which was to be cut through and the ends closed with tight ligatures. In this way we hoped to repeat, in the thorax, what Doctor Allen had already accomplished in the abdomen by the gradual obliteration of the aorta below the renals. This he had done in two or three stages with our modification of the Halsted aluminum bands, following which the aorta was divided through the obliterated segment, between sutures. A number of dogs died from premature cutting of the aortic walls by the bands, but the occlusion succeeded often enough to prove that the complete division of the aorta below the renals could be made effective by gradual occlusion, provided the bands were removed in time to anticipate the perforation and fatal hemorrhage, which occurred more often after the seventh or tenth day. The dangers of the metallic compression led me to try the method of occlusion by plicating the thoracic aorta which, with Doctor Allen's able assistance, I tried, at irregular intervals, for two and one-half years (1910-1913), during which the operation was performed on 151 dogs. Seventy dogs survived the first plication; 73 the second, and 11 reached the third or obliterative plication. But it was found that when the infolded walls filled the lumen of the aorta, the force of the systolic wave stretched a passage for a narrow blood stream beyond the seat of the obstruction. While no dogs survived the third plication, and they did not live long enough to permit a complete *dry* section of the plaited segment, the experiments demonstrated the capacity of the heart and collateral vessels to adjust themselves to an extreme degree of aortic stenosis which would have been fatal if the artery had been suddenly closed by a total ligature.

As the defects and failures of all previous methods of gradual occlusion have been fully brought out by Doctor Pearse, I will not consume more time with further commentaries on past or historic procedures. I am glad, however, to avail myself of this opportunity to submit to your consideration a new and unpublished method of gradual occlusion of the aorta, both abdominal and thoracic (outside of the arch), which promises to deliver the long sought procedure of a gradual and safe aortic occlusion, at least as this has been found effective in dogs and, now, holds a good prospect that it will be found equally feasible in man. For this privilege, I am indebted to Dr. James C. Owings, a young and capable experimenter—assistant to Dr. Harvey B. Stone in the Surgical Hunterian Laboratory at Baltimore—who has just been mentioned by Doctor Stone in connection with his experiments with heparin.

Through Doctor Owings' personal letters, I have learned of his successful efforts to obtain a gradual but complete occlusion of the abdominal and thoracic aortas, in practically any part of their course, by means of a rubber band which is made to conform to the shape of an hour-glass. The band, acting as a ligature, is narrowed progressively in three operations at intervals of about three weeks each. Wide stationer's rubber bands for the preliminary compression were chosen because they give with the pulsation of the vessel, to a certain extent, and are, therefore, less likely to cut through than metallic bands. The rubber band is plicated into the shape of an hour-glass so that there is no abrupt change in the caliber of the vessel, thereby eliminating the sharp edge against which the vessel wall would otherwise be constantly butting.

It has been necessary to apply three of these bands at three successive operations, at three weeks' intervals, before sufficient collateral circulation has

been developed to make ligation possible. When the third stage is reached, the vessel is doubly ligated with braided silk and the intervening segment is thoroughly crushed with Kelly forceps, to promote thrombosis and fibrosis. The ligatures are separated from each other by about 1.5 cm., after which the crushed segment is cut between ligatures. On April 1, 1940, Doctor Owings wrote that he had four dogs ready for this final step. One of these had been tied off for nearly a year and the others about seven months. The rubber bands are applied at three-week intervals; one below the other, or, occasionally, one over top of the other. This is necessary to produce sufficient collateral circulation because the yield of the ligature plus the atrophy of the aortic wall is enough to reestablish the pressure below the ligature within about three weeks and, if it is not again compressed at least twice, the animals die, from insufficient circulation to the kidneys and liver, when the final occlusion is made.

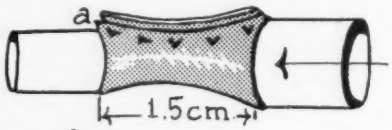

Fig. 1

Fig 2

Fig 3. (a)


Fig 3 (b)

FIG. 1.—(a) Cut edges of rubber. One edge is sometimes left long and folded over the short edge to make it smooth.

A rubber band is placed around the aorta and fixed in position with mattress sutures. Tension is judged by a flabby vessel below, with no pulsation and only a faint thrill to be felt.

FIG. 2.—Longitudinal section to show what a guess it is to estimate the degree of constriction. Note that segment above band is much larger than below. A band is placed just above an intercostal vessel to keep it from slipping down.

FIG. 3.—(a) Aorta tied and segment between ties crushed with a Kelly clamp to induce thrombosis and fibrosis. Note that upper and lower segments, at this stage, are nearly the same caliber, because there is very little tension in either of them. The ties are of heavy braided silk. It is usually necessary to apply three bands, either one immediately below the other, or one over top of the other at three- or four-week intervals before sufficient collateral circulation can be developed, because tension returns by atrophy of the vessel wall and relaxation of the bands.

FIG. 3.—(b) Final stage: Showing the crushed and obliterated segment of aorta divided.

In dealing with the thoracic aorta, the rubber bands were applied to the descending portion below the arch. He has made no attempt to obliterate the vessel proximal to the arch. The surgical approach has always been transpleurally by an anterior thoracotomy.

On May 21, 1940 (after the meeting of the American Surgical Association at St. Louis) Doctor Owings wrote: "Since my last letter I have cut across the aorta below the point of constriction, ligating the two ends with ordinary black silk and allowing them to retract. All three animals have done very well. In fact, they were up and eating within a few hours after the operation.

"I am going to make some studies on kidney function and try to get roentgenograms of the collateral circulation before the animals are sacrificed, and hope to report the work in further detail before the Southern Surgical Association, next fall."

In order to give a still further conception of Doctor Owings' procedure, he has, obligingly, made four diagrammatic sketches which I am reproducing with their appended legends (Figs. 1, 2 and 3a and b).

No one who is familiar with this type of experimental work can fail to be

impressed with the simplicity and soundness of Doctor Owings' procedure and the very extensive field of surgical application which it suggests, since it is applicable to the whole extent of the aorta from the arch to the bifurcation.

DR. HERMAN E. PEARSE (Rochester, N. Y., closing): Several questions were asked me concerning the cellophane that was wrapped around the vessels. It was ordinary DuPont cellophane, No. 300 P.T., soaked in alcohol or mercury oxycyanide, folded to two or four thicknesses, making a strip about 1 cm. wide and wrapped loosely twice or three times around the vessels. The ends were tied or were sutured with silk, to hold them from slipping.

As to Doctor Reichert's question, I must confess my ignorance. I know of some of Doctor Van Allen's publications, but I did not believe that he had used cellophane.

It is possible to divide the thoracic aorta completely. I have done it after thrombosis with the spring. The reaction of the dog's thoracic aorta is exactly comparable to that of many human arteries, and I believe this should always be kept in mind in experimental work on the subject.

DR. FRANK LAHEY (Boston, Mass.): How does cellophane cause this reaction?

DOCTOR PEARSE: I do not know. It is a terrific reaction, long-continued and persistent but not associated with ordinary inflammation. There are very few polymorphonuclear leukocytes in the tissue. There are many large phagocytic cells often containing fragments of cellophane, which would lead one to believe that it is a foreign body reaction.

THE PREVENTION OF ISCHEMIC GANGRENE FOLLOWING SURGICAL OPERATIONS UPON THE MAJOR PERIPHERAL ARTERIES BY CHEMICAL SECTION OF THE CERVICODORSAL AND LUMBAR SYMPATHETICS*

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THE PREVENTION of ischemic gangrene following occlusion of the major peripheral arteries has been studied since the time of Petit who, in 1731, observed the local effects of ligation of an artery. The results of occlusion of the major peripheral vessels have been extensively investigated, both clinically and experimentally. These studies have resulted in a fairly complete understanding of the underlying pathologic physiology. Following major peripheral arterial occlusion certain changes occur, many of which are commonly accepted, whereas some are considered speculative. One factor common to all major arterial occlusions is a decrease in blood supply distal to the occlusion, resulting in ischemic changes varying from necrosis of a few cells to massive gangrene of an entire extremity.

The prevention of ischemic gangrene following sudden occlusion of a major peripheral artery is desirable in all cases. The causes of this most undesirable complication and the methods of its prevention will be discussed in this presentation. The lesions of the peripheral vascular tree which are potential or actual causes of ischemic gangrene, in order of frequency are: traumatic injuries, aneurysms, and arterial embolism. The usually employed surgical treatment in arterial injuries and aneurysms is sudden obliteration of the involved artery either by ligature or by suture; whereas in arterial embolism, arteriotomy and arteriorrhaphy have been the so-called procedures of choice.

The peripheral arterial system is composed of a main artery with secondary branches (collateral circulation), capillaries (both arterial and venous), venules, and main collecting venous trunks. The vessels as far as the capillaries are neuromuscular tubes through which the blood circulates to supply the component parts of an extremity with both nourishment and oxygen. The veins are the disposal system which returns the used blood back to the heart and lungs. A third circulation is the lymphatic system which is dependent upon an intact vascular system for its normal function. The blood vascular system, with the exception of the capillaries, is under the influence of the sympathetic nervous system, which controls the caliber of the vessels and also to a certain degree the blood volume flow. Three essentials for a

* Read before the American Surgical Association, St. Louis, Mo., May 1, 2, 3, 1940.

normal arterial blood volume flow are: elasticity of the vessel wall, vasomotor balance, and cardiac output. The venous circulation is dependent upon vasomotor control, capillary pulsations, and a normal peripheral venous pressure (Kountz[1]). In Kountz's perfusion experiments it was almost impossible to force the blood through the capillary bed when the veins had been previously emptied. However, with a tourniquet around the limb, elevating the venous pressure 10 to 20 Mm.Hg., a maximum arterial flow occurred. From this investigation he concluded that peripheral venous pressure has an important influence on the passage of blood through the capillaries. According to him, Silbert, Lilienthal, Collens and Wilensky observed the same phenomena clinically in peripheral vascular diseases. Therefore, it is necessary that a certain residual pressure be present both on the arterial and venous sides to maintain normal peripheral capillary circulation.

That the third circulation, or lymph flow, is dependent upon the normal blood flow (arterial and venous residual pressure plus capillary pulsation) has been demonstrated experimentally by Monterio,[2] and McMasters and Parsons.[3] McMasters and Parsons, and Cressman and Blalock[4] showed experimentally that capillary pulsations were necessary for the lymph flow. Monterio demonstrated that by increasing the peripheral residual pressure by sympathectomy there occurred an increased lymph flow in the extremities. Therefore, we are justified in assuming that a normal peripheral circulation (arterial, venous, and lymphatic) is dependent upon a residual arteriovenous pressure, capillary pulsations, and sympathetic balance. Any alteration in arterial or venous pressures, blood volume flow, or sympathetic control will produce marked disturbances in normal function. When these become irreversible, permanent tissue damage results.

It is well known that ischemic gangrene does not always follow occlusion of a major peripheral artery. The necrosis is prevented in such an instance by the maintenance of an adequate blood supply through the collaterals. In both the upper and lower extremities there is an abundant collateral circulation which never functions to its full capacity as long as the main vessels function normally. It is only when the main artery is occluded that the collaterals take over the function of the occluded vessel. The functional capacity of the collateral circulation to compensate for major arterial loss varies according to site of obliteration, *i.e.*, there are certain anatomic locations in which the main arterial circulation is most vulnerable, due to a deficient collateral vessel anastomosis. The obliterations of the common femoral, carotid at its bifurcation, and the popliteal arteries are frequently followed by grave consequences. The collateral circulation around the shoulder, elbow, hip, hand, and foot is very abundant (Figs. 1, 2 and 3). The collaterals around the knee are rather scant because there are no great masses of muscle bridging the knee joint (Sehrt[5]).

The function of the collaterals is to maintain adequate blood supply to the tissues of the body. In reality they are nothing more than arterial branches that originate from the main parent arterial stem. They, in turn,

break up into an extensive capillary network in the bone, muscle, subcutaneous tissue, and skin. They are under control of the same sympathetic system which controls the main artery. Therefore, any disturbances, direct or reflex, within the main arterial stem affect the collaterals secondarily. Probably the

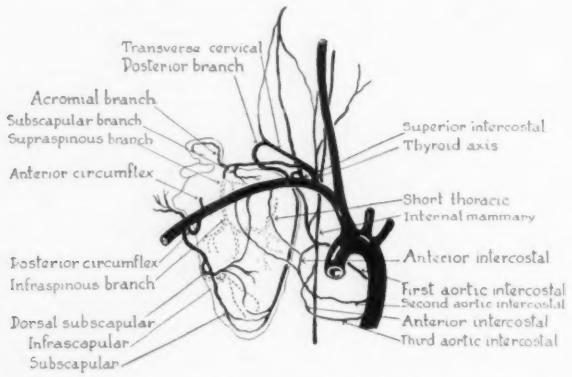

FIG. 1.—Illustrating the extensive collateral circulation extending from subclavian to the upper extremity. Figure 1 blends into Figure 2.

most important function of the collateral vessels is the substitution of an efficient circulation to an extremity in which the main stem artery has been obliterated. In fact, according to Matas,[6] the important results for the successful obliteration of the main artery of an extremity are dependent upon

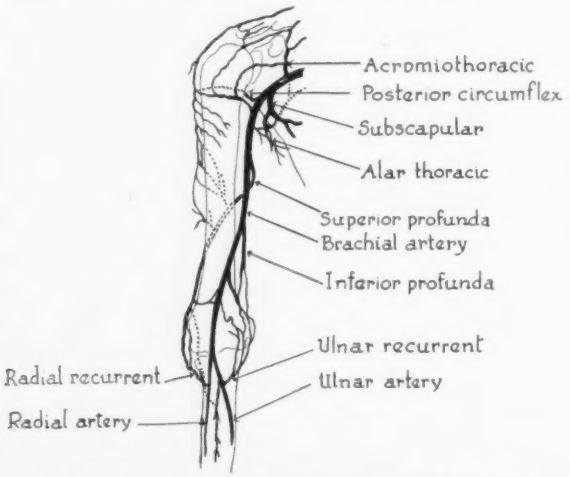

FIG. 2.—Drawing showing the collateral circulation bridging the axillary to brachial. The illustration also demonstrates the collateral vessels of the entire upper extremity.

the efficiency of the collateral circulation. It is, therefore, an indubitable fact that the incidence of ischemic gangrene following sudden occlusion of a major artery of an extremity is dependent upon the inadequacy of blood volume flow through the collateral vessels, and varies from 5.2 per cent[7] to

45.8 per cent.[8] The incidence varies greatly in the different types of obstruction. In traumatic lesions the incidence of ischemic gangrene varies from 11 per cent[9] to 45.8 per cent;[8] in ligations for aneurysms, 5.2 per cent[7] to 15 per cent;[10] and in embolic obstruction it is probably highest of all, averaging over 30 per cent.[11, 12]

In traumatic lesions of the main arterial trunks of the extremities, the incidence of gangrene, according to Tuffier,[13] is 40.2 per cent. Heidrich[14] reported an incidence of 15.5 per cent of ischemic gangrene in 995 cases following ligation of the large peripheral arteries. Kretzschmann[9] reported an incidence of 11 per cent gangrene requiring amputation in a series of 72 cases of traumatic lesions involving the peripheral arteries. Makins,[8] in 101

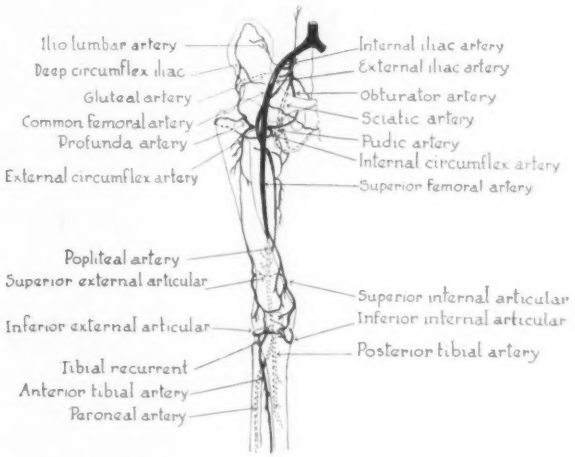

FIG. 3.—Drawing showing the abundant collateral circulation bridging the iliacs to the femoral. The collateral circulation bridging the popliteal is also illustrated.

cases of war wounds of the major peripheral arteries, had an incidence of 28 per cent ischemic gangrene. Sehrt,[5] in 1938, stated that ligation of the main artery in the lower extremity resulted in gangrene in 20 per cent, whereas ligation of the axillary artery resulted in ischemic gangrene in only 7.8 per cent. That the incidence of ischemic gangrene varies following occlusion at different levels of the same artery as well as the different major arteries of the extremities, is demonstrated by the following statistics: subclavian, 25 per cent; axillary, 16.6 per cent; brachial artery, 23 per cent; femoral, 25 per cent; popliteal, 41.66 per cent; tibial, zero per cent; carotid, 33.33 per cent (Makins[8]). Makins stated that there was considerable variation in the incidence of gangrene following injury to the popliteal artery according to the location of the arterial injury. In 15 cases with injuries in the upper third, gangrene occurred in three (20 per cent); in 25 cases with injuries in the middle third, gangrene occurred in ten (40 per cent); and in 20 cases with injuries in the lower third, gangrene occurred in seven (35 per cent). These significant variations in the incidence of gangrene at

various levels of the popliteal are dependent upon the anastomosis about the knee. Tuffier[13] reported an incidence of 41.16 per cent gangrene following injury to the popliteal artery in 24 cases, and stated that, in general, ligation of the arteries at the roots of the limbs results in ischemic gangrene in 40.2 per cent.

In the majority of traumatic lesions of the peripheral arteries, the preferable treatment is the ligation of the injured vessel, which results in its sudden occlusion. The injury plus the sudden occlusion produces pathologicophysiologic changes that are responsible for the high incidence of ischemic gangrene. The rapid progression of this process also prevents the development of an adequate circulation. Both the pathologicophysiologic and collateral developments will be discussed later.

Sudden arterial occlusion by an embolus produces an incidence of ischemic gangrene comparable to the incidence occurring following traumatic arterial lesions. Both produce sudden occlusion and the pathologic physiology is very similar. Danzis[11] reported 129 cases of arterial embolectomy with an incidence of gangrene in 39.5 per cent. Linton[12] collected 282 cases of arterial embolectomy, in only 30 per cent of which was there a return in circulation. In a personal series of 44 emboli occurring in 36 patients, of whom only 12 were operated upon, the incidence of ischemic gangrene necessitating amputation was 67 per cent. In another series of 17 cases, treated symptomatically, ischemic gangrene developed in 12, or 70 per cent. In arterial embolism, as in ligation for arterial trauma, there is a sudden occlusion of the main artery of an extremity without sufficient time for the collateral circulation to develop, with the result that the high incidence of ischemic gangrene is due to failure of the peripheral circulation.

In contrast to the above is the low incidence of ischemic gangrene following sudden occlusion of the major artery of an extremity for the cure of aneurysm. Matas[7] reported an incidence of 5.2 per cent in 154 cases of aneurysm. Bird,[10] on the other hand, estimates that the incidence of gangrene following occlusion of the popliteal artery for aneurysm varies from 8 to 15 per cent. The low incidence following treatment of aneurysms is due to the fact that: (1) the aneurysm results in an interference with the normal blood volume flow, which is more marked in arteriovenous aneurysms than arterial; and (2) there is sufficient time for the estimation of the collateral efficiency and the development of the collateral circulation in cases in which it is found deficient. This contention is demonstrated by De Fourmestraux's[15] statistics as follows: in ligating the common carotid for hemorrhage, the mortality was 54 per cent; for tumors, 46 per cent; for arterial aneurysms, 13.5 per cent; and for arteriovenous aneurysms, 7 per cent—demonstrating also that the arteriovenous fistula develops a more abundant collateral than the arterial type.

The sudden occlusion of a major peripheral artery produces a typical syndrome which is familiar to the majority of surgeons. This is characterized by pain and sudden cessation of the peripheral pulse, followed by various

color changes in the skin, lowering of the surface temperature, numbness, and varying degrees of functional loss.

According to Pearse,[16] there is a definite elevation in arterial pressure proximal to the point of occlusion, the height of the pressure varying with the caliber of the vessel occluded. There was a marked fall in arterial pressure distal to the ligature in all of his experiments. He stated that this fall in systolic pressure never returned to normal. Wilson,[17] in a large series of well controlled experiments, reported some interesting findings following the occlusion of the main artery to an extremity. Following ligation of the main artery of the hind leg of the rabbit, the carotid pressure rose from 138 to 144 Mm.Hg., whereas the arterial pressure distal to the ligature fell from 128 to 78 Mm.Hg., a drop of 50 Mm. The venous pressure dropped from a normal of 6 to 3.8 Mm.Hg. A fall in peripheral venous pressure is a very serious handicap to the reestablishment of blood volume flow, as demonstrated by Kountz[1] in his perfusion experiments upon amputated lower extremities. He stated that when the veins were emptied before amputation it was almost impossible, by perfusion, to force modified blood through the capillaries. However, on elevating the venous pressure 10 to 20 Mm.Hg. by applying a constrictor, normal blood flow was established. The arterial volume flow per minute fell from 95.60 to 30.46 cc. There also occurred a cessation of arterial pulsations distal to the occlusion. It is seen from these experiments that both arterial and venous limb pressures fell markedly and that the mass of blood in the extremity and the volume flow of blood per minute were greatly diminished. He further demonstrated that the oxygen consumption per minute of the tissues of the whole limb and of the tissues below the obstruction was diminished. Mulvihill and Harvey[18] demonstrated experimentally that ligation of a major peripheral artery resulted in the decrease of from 10° to 30° F. temperature in the involved extremity. However, a return of the temperature of the extremity to its previous level occurred in about 13 hours after ligation of the main artery to the limb. They believed that this return of the temperature to normal was a vasomotor phenomenon, which they later proved by blocking the sympathetic vasoconstrictor fibers. Theis[19] demonstrated that there is a definite fall in arterial pressure following ligation of the femoral artery in the dog. The blood pressure remained 10 to 30 Mm.Hg. lower distal to the arterial occlusion and it failed to return to normal at any time during a seven months' observation. The results of Haimovici's[20] experiments explain the phenomena resulting from the sudden occlusion of a major peripheral artery. This author introduced a balloon into the major peripheral artery of a dog's extremity. It was possible to distend the balloon to variable pressures. He was able to partly occlude the vessel, occlude the vessel slowly, or produce a sudden complete occlusion of the vessel. He observed that there was no vasomotor reaction if the vessel was incompletely obliterated and similarly no reaction occurred if the lumen was slowly obliterated. However, if sudden complete occlusion was produced, a marked vasomotor response occurred. This response

consisted of vasospasm in the region of the obstruction, vasospasm in the distal vessel, and arterioles and even the capillaries were affected. He also stated that this vasospasm could extend to the collaterals and manifest itself in the neighboring vessels even at a distance from the occlusion. Haimovici[20] called this vasomotor reaction "arterial colic," and refers to the pain accompanying vasoconstriction of the vessels as such. Haimovici's experimental observations are confirmed clinically by the manifestation in arterial occlusion by embolism and thrombosis. If the arterial embolus is not occlusive but secondary thrombosis slowly takes place, practically no vasomotor disturbances occur. This is because vasoconstrictor impulses affecting the collaterals are not initiated, and, also, because there is adequate time for the development of a collateral circulation. Conversely, if the lumen is suddenly occluded by an embolus, vasoconstrictor impulses originate at the site of the embolus and spread to all the arterial ramifications including the collaterals; and the process progresses so rapidly that there is insufficient time for the development of collaterals. It is evident that both experimental and clinical investigations are comparable and substantiate each other. Therefore, the major symptoms following occlusion of a peripheral artery are dependent primarily upon the cessation of blood volume flow through the artery distal to the ligature and secondarily upon the initiation of vasospasm which may spread to the entire vascular tree as described by Haimovici.[20]

It is common knowledge that trauma to a large peripheral artery produces vasospasm at the site of the trauma which not infrequently spreads to the whole vascular tree. We have seen several cases of segmental arterial spasm with concomitant involvement of the distal vascular tree associated with perivascular trauma resulting from a so-called "*Metzger Verletzung*" (butcher's injury). This injury occurs during the skinning of beef, and is the result of slipping of the knife, resulting in a stab wound on the upper medial aspect of the thigh and trauma either of the femoral vessels or the perivascular tissues in the upper end of Hunter's canal. Even though the femoral vessels are not divided in many instances, reflex vasospasm is of such magnitude that the clinical manifestations are identical with those of complete arterial section. Montgomery and Ireland[21] collected 42 cases of traumatic segmental arterial spasm and added two cases of their own. We have, also, observed two cases of segmental arterial spasm of the brachial artery following fracture of the humerus. Following the injury the arm distal to the segmental arterial spasm and the forearm, including the hand, were cadaveric. There was no pulsation in the radial, ulnar, or brachial arteries distal to the injury. True vasospasm was proven by the immediate relief following novocain analgesia of the stellate ganglion, which produced an immediate return of color to the arm and pulsation in the distal arteries.

Halsted,[22] Leriche,[23] Matas,[24] and La Roque[25] demonstrated, clinically, that spastic contraction of an artery at a point distal to the site of trauma incidental to handling large blood vessels occurs not infrequently. According to them, this spasm was the result of vasomotor response to stimuli.

Matas[24] reported a case of temporary obliteration of the pulse distal to an aneurysm in which, at operation, a patulous lumen of the artery was demonstrated. According to Leriche,[23] ligation of a major peripheral artery initiates a vasomotor spasm of the vessels of the extremity, and because of this he advised double ligation with severance of the vessel between the ligatures instead of ligating in continuity.

We believe that there are varying degrees of venospasm occurring concomitantly with arterial spasm. The reverse has been proven both experimentally and clinically, *i.e.*, that in sudden occlusion of the deep major vein there occurs an associated arterial spasm which at times simulates arterial embolism. Uggeri and Massone[26] reported three cases of thrombophlebitis of the extremities with ischemic arterial manifestations. They divided the arterial ischemic reactions into three groups: Group I: The ischemic symptoms appear with or after the phlebitic signs. Group II: The signs of circulatory deficiency appear suddenly and to such a degree that it is difficult to differentiate them from those produced by primary arterial embolus. Group III: There are the same clinical manifestations as in Group II, except that the onset and the ischemic manifestations are less intense. As the arteries reveal practically no pathologic changes, the ischemic manifestations are due to vasospasm initiated in the thrombophlebitic lesion. DeBakey, Burch, and Ochsner,[27] in a series of experiments upon dogs, isolated a segment of the femoral vein between two ligatures, aspirated the blood from the segment, and injected an equal quantity of 40 per cent aqueous solution of sodium salicylate. They found that the peripheral volume pulsations decreased (52.5 per cent) following ligation of the vein, reaching a maximum within five minutes. When sodium salicylate was injected into the isolated segment producing a chemical phlebitis, the peripheral volume pulsations decreased (51.6 per cent). This decrease in the peripheral arterial pulsations did not occur if a previous sympathetic ganglionectomy had been performed. Conclusions deducted from their experiments in determining the possibility of an ipsolateral arterial and arteriolar vasoconstriction in femoro-iliac thrombophlebitis are as follows: "It is evident that a chemical irritant placed either in the lumen of the main vein of the extremity or in the perivascular tissue of this vein produces a marked diminution in the volume (51.6 per cent) of peripheral pulsations. However, interruption of the nerve pathways by local infiltration with procaine hydrochloride at the site of the chemical irritation or by resection of the lumbar sympathetic ganglia and chain abolish this effect. This would suggest, therefore, that the decrease in volume pulsations following chemical phlebitis and periphlebitis is due to vasoconstrictor impulses initiated locally by the chemical irritant and coursing through the sympathetic ganglia in order to reach the terminal arterial vessels of the extremity." Ochsner and DeBakey[28, 29] also demonstrated that in clinical cases of femoro-iliac thrombophlebitis (phlegmasia alba dolens) there is marked decrease in the ipsolateral peripheral arteriolar pulsations which is probably responsible for the white color and decrease in surface temperature

in these cases. The ischemic manifestations in these can be abolished effectively by novocain block of the regional sympathetic ganglia. They believe that the manifestations in thrombophlebitis are due less to the associated venospasm than to a reflex arteriospasm.

Another circulatory disturbance to which we attach great importance is the interference of blood flow through the vasa vasorum in arterial spasm. As the vasa vasorum are the channels through which the vessel wall receives its nourishment, and as they are in juxtaposition to the musculature, we believe that in arterial spasm there is marked interference with the blood supply to the arterial wall. According to Cummins,[30] even the endothelial lining of the arteries receives some of the nourishment from the blood carried in the vasa vasorum. It is likely that interference with nutrition to the endothelium even for a short interval will cause sufficient pathologic cellular changes in the intima to produce arteriolar thrombosis.

From the above discussion, it can be concluded that a sudden occlusion of a major peripheral artery produces the following pathologico-physiologic changes: (1) sudden obliteration of the peripheral pulse; (2) marked decrease in blood volume flow; (3) rapid fall in the temperature of the limb tissues; (4) temporary or even permanent cessation of capillary pulsations; (5) marked and sustained decrease in the arterial and venous residual pressures; (6) moderate to severe vasospasm of the entire arterial tree distal to the arterial obliteration; (7) decrease or cessation of lymph flow; (8) concomitant venospasm; (9) mass of blood in the limb and blood volume flow per minute greatly diminished; (10) interference with vasa vasorum circulation by the arterial vasospasm; and (11) pathologic changes within the vessel wall resulting in thromboses. Unless the above pathologic physiology is prevented, or immediately corrected after the first clinical manifestations, ischemic gangrene, either segmental or massive, will develop and this may result in the loss of an extremity or, not infrequently, life itself.

In surgery of the major peripheral arteries, all of the above changes must be constantly kept in mind and methods must be instituted preoperatively and postoperatively to prevent their occurrence. A failure to appreciate these fundamental principles will only result in disaster.

To prevent the development of ischemic gangrene following surgery of the main peripheral arteries (which in the majority of instances consists of obliteration of the vessel by ligature), it is necessary to: (1) test the efficiency of the collateral circulation; (2) develop the collateral circulation when found deficient; (3) prevent segmental and diffuse arterial vasospasm; (4) prevent venospasm; (5) increase the blood volume flow through the collaterals and the main vessel distal to the ligature; (6) maintain capillary pulsations; (7) maintain lymph flow and spread; (8) increase peripheral residual pressure; (9) maintain a normal or elevated tissue temperature; (10) increase blood volume flow through the vasa vasorum; and (11) prevent thrombosis of peripheral arterioles and capillaries.

In testing the collateral circulation the following methods have been ad-

vocated: Matas compressor; Moszkowicz' test; oscillometric readings; plethysmographic readings; and thermocouple readings. We are partial to the Matas compressor test because it produces occlusion of the involved artery, shunting the blood flow through the collateral vessels. In other words its application is identical to a temporary ligature without damage to the artery. The test is easily performed and gives accurate information as regards the efficiency or inefficiency of the collateral circulation during obliteration of the main peripheral artery. Moszkowicz's test is mainly used to determine the site of amputation in the presence of peripheral gangrene and is not applicable to the subject under discussion. The other tests are important in determining the presence or absence of as well as the degree of vasospasm, but they do not give reliable information concerning the collateral efficiency, because the main vessel is patent. However, when used in conjunction with the Matas compressor they are invaluable because the degree of vasospasm can be accurately determined following the obliteration of the main peripheral artery by the compressor. The above test can be used only in elective surgery of the large peripheral arterial trunks. In the elective cases there is ample time for prolonged study, and all the information regarding collateral circulatory efficiency, vasospasm, venous presure, residual arteriovenous pressure, tissue pressure, and tissue temperature can be obtained with safety, preoperatively. However, in traumatic lesions of the major peripheral arteries and in sudden occlusion by an embolus, the time element precludes accurate study and testing of the efficiency of the collateral circulation. In the vast majority of traumatic lesions the patient is in shock; therefore, many of the tests advocated above would be contraindicated. Even if they were used, the results would be of little significance because experimental results and clinical observations have demonstrated that in the traumatic lesions as well as in sudden and complete embolic occlusions, the collateral circulation is inadequate due to the pathologic physiology induced by vasospasm. Therefore, methods must be employed that will establish and maintain an efficient collateral circulation, increase the blood volume flow, and at the same time prevent arteriovenous vasospasm of the peripheral vascular tree. As the incidence of gangrene is in direct proportion to the efficiency of the collateral circulation, the methods employed to prevent ischemic necrosis are mainly those of developing an adequate and sustained blood volume flow through the primary and secondary collateral vessels.

The methods used to develop the collateral circulation are divided into the following groups: (1) spontaneous; (2) mechanical; and (3) physiologic. The spontaneous development of a collateral circulation is the result of incomplete and gradual interference with the blood flow through the main peripheral artery. This commonly occurs in peripheral aneurysms, of which the arteriovenous type is likely to develop a more efficient collateral circulation than the arterial. This is because an arteriovenous aneurysm in the majority of instances produces a greater interference to the arterial blood flow at the site of the fistula. The obstruction to the blood flow by an arterial

aneurysm depends to a great extent upon the type and saccular form of the aneurysm. However, a good preoperative collateral circulation in an aneurysm is no warranty that it will be maintained postoperatively. The statistics of Matas,[7] Bird [10] and De Fourmestraux[15] demonstrate that spontaneous development of the collateral circulation is common in aneurysms.

The mechanical methods used to develop the collateral circulation in order of their efficacy are: the Matas compressor; intermittent venous occlusion (including the Pavaex); and simultaneous ligation of the concomitant vein. The Matas compressor obliterates the main artery above the arterial lesion shunting all the peripheral circulation through the collaterals distal to the point of obliteration. The compressor is applied daily for increasing intervals of time until an adequate collateral circulation has been established. The time required to obtain the desired results is occasionally considerable and, therefore, can be used only in the elective cases of vascular lesions. It cannot be used as an emergency procedure. Therefore, it is of little value as a preparatory method in traumatic lesions and peripheral arterial embolism.

The intermittent venous occlusion and the passive vascular exercises have the same limitations of application as the Matas compressor as a preoperative method in all but the elective cases. Both of these mechanical methods are more frequently used in the presence of an inadequate collateral circulation in progressive organic disease of the peripheral arteries. However, they might be used postoperatively to advantage, if the collateral circulation manifests evidence of beginning failure.

Simultaneous ligation of the concomitant vein when a large peripheral artery is ligated has been used rather extensively over a period of years in an attempt to increase the blood volume flow through the peripheral capillaries. Von Oppel[31] was probably the first to advocate ligation of the concomitant vein when a large peripheral artery was ligated. However, Makins[8] states that it was possible that John Hunter ligated the accompanying vein in his first three cases of ligation of the femoral artery for aneurysm. Since von Oppel's first report, considerable clinical and experimental data have been presented in favor of and against the procedure. Von Oppel[32] ligated the popliteal satellite vein simultaneously with the popliteal artery in six cases of peripheral vascular disease. He noted a marked change in the color of the foot corresponding to an increased collateral circulation. He believed that the vein ligation inhibited to a certain degree the incidence of ischemic gangrene. Holman[33] states "that the evidence, amid the welter of many inconclusive experiments, proves beyond doubt the beneficial effect produced upon the nutrition of a limb by the simultaneous ligation of artery and vein." Tuffier[13] recorded an incidence of ischemic gangrene of 41.66 per cent following ligation of the popliteal artery and an incidence of 40.2 per cent following ligation of all large peripheral arteries at the root of the limbs. In cases in which there was simultaneous ligation of artery and vein (popliteal), the respective incidences were 21 per cent and 24.5 per cent, almost a 50 per cent reduction. Heidrich[14] reported a series of 198 arterial ligations with simultaneous liga-

tion of the satellite vein with an incidence of only 8.5 per cent gangrene. Whereas, in 995 cases in which the artery alone was ligated, the incidence was 15.5 per cent. Makins[8] reported a 28 per cent incidence of gangrene in 101 cases in which the artery alone was ligated and a 19.7 per cent incidence in 71 cases with simultaneous ligation of the artery and the vein. Brooks,[34] after extensive experimental work, concluded that this procedure is of immediate benefit in decreasing the incidence of gangrene, but cautions against the late effects in promoting venous stasis. He advocated ligation of the concomitant vein when the common femoral, common carotid, and popliteal arteries were ligated, because occlusion of these arteries is frequently followed by gangrene. According to Coudray,[35] ligation of the internal jugular vein simultaneously with the carotid diminishes the risk of hemiplegia. Sehrt[5] reported an incidence of 20 per cent gangrene following ligation of the main peripheral artery of the lower extremity. However, when the vein and artery were ligated concomitantly the incidence fell to 9 per cent. In a very excellent and well-controlled experimental investigation of the effects of simultaneous ligation of major peripheral arteries and veins, Wilson[17] found that when the femoral artery was ligated the peripheral arterial pressure dropped from 138 to 78 Mm.Hg., the venous pressure dropped from 6 to 3.8 Mm.Hg., and the blood volume flow per minute fell from 95.60 to 30.46 cc. When artery and vein were simultaneously ligated the arterial pressure dropped from 138 to 94 Mm.Hg., the venous pressure was elevated from 6 to 22 Mm.Hg., and the blood volume flow per minute decreased from 95.60 to 25.81 cc. Wilson concluded from his experiments that the simultaneous ligation of the concomitant vein does not lessen the incidence of ischemic gangrene which follows the ligation of the main artery of a limb, nor does it decrease the severity of or the distribution of muscle necrosis.

From the above statistics, it can be concluded that the simultaneous ligations of major peripheral arteries and veins diminishes but does not prevent the occurrences of ischemic gangrene. Wilson's investigations demonstrated that such a procedure causes a marked reduction in blood volume flow, which is undesirable. We believe that the procedure is not indicated because better results can be obtained by abolition of sympathetic impulses. Also as shown by DeBakey, Burch, and Ochsner[27, 36] simple ligation of the femoral vein of the dog produces a reduction of volume pulsations (52.5 per cent) in the foot. Although various methods have been used to decrease the incidence of ischemic gangrene following occlusion of major peripheral arteries, none has been universally successful. Because of the occasional failure in maintaining adequate circulation in such cases by means of the usually employed methods and because of the invariably good results which we have obtained from the use of physiologic method, *i.e.*, interrupting the vasoconstrictor impulses, we have abandoned all other methods in favor of this one. It is a method that can be used preoperatively, at the time of operation, and postoperatively, in all operations upon the peripheral vascular system. It should be designated the

physiologic method because those impulses (vasoconstrictor) which are responsible for the diminution of the blood are prevented.

Jaboulay,[37] in 1899, proposed periarterial sympathectomy to increase the circulation in the extremities. Leriche,[38] in 1913, performed periarterial sympathectomy upon the femoral artery and observed that the maximum effect occurred within 36 hours postoperatively. The effect was an increase in the arterial pressure "which usually became an hypertension," and also an increase in surface temperature. These effects, however, gradually disappeared. Leriche, in 1913, stated that "the clinical phenomena following pariarterial sympathectomy are in accordance with the experimental facts determined by Claude Bernard in 1832." Herrick, Essex, and Baldes[39] demonstrated experimentally that there was an increased blood volume flow through the femoral artery of the dog following lumbar sympathectomy. They found that the

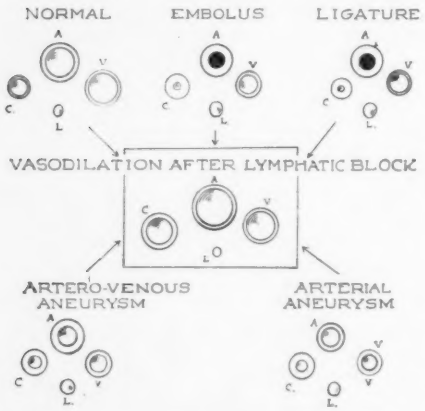

FIG. 4.—Drawing illustrating the effect of sympathectomy upon various occlusive conditions of the major peripheral vessels.

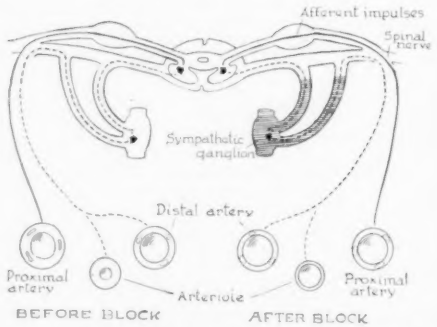

FIG. 5.—Demonstrating the effect upon the normal vessels and vasa vasorum following sympathetic block.

increased flow was twice that of normal. They also observed at the same time that ether anesthesia produces an increase in blood flow equal to that produced by sympathectomy. The latter observation is of great significance because in ligating a peripheral artery the circulation may seem adequate at the time of the operation only to fail several hours later, the vasodilatation being due to the anesthesia.

Mulvihill and Harvey[18] observed that there occurred constantly a fall in temperature of the extremity following ligation of the dog's iliac artery. However, such a decrease in temperature was obviated by the simultaneous performance of sympathectomy. On the basis of his experimental observations, Theis[19] found that following sympathectomy, there was an increased blood volume flow through the collateral arterioles as well as an increase in both surface and deep temperatures. He also demonstrated that in the main artery below the ligature there was an increased blood volume flow of 60 volumes per cent, and a 15 per cent average rise in blood pressure. The author observed that the fall in the peripheral arteriole pressure upon the

sympathectomized side rapidly returned to normal and at the end of ten months was the same as the normal blood pressure. His conclusions were that sympathectomy preceding the ligation of the femoral artery in the dog resulted in an increased blood volume flow, sustained normal peripheral pressure, prevention of arterial vasospasm, and the immediate and rapid development of an adequate collateral circulation. This marked increase in collateral circulation remained constantly greater than the physiologic increase taking place in the unsympathectomized limb. This is demonstrated graphically in Figure 5 which demonstrates the effect of sympathectomy upon the normal peripheral vascular tree. Monterio[2] has shown, experimentally, that there is a definite increase in the lymph flow of an extremity following lumbar sympathectomy. This is an important observation because in the absence of peripheral pulsations (produced by arterial occlusion and vasospasm), McMasters and Par-

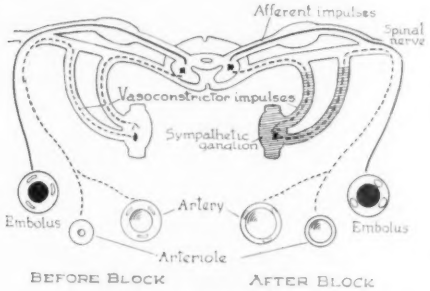

FIG. 6.—Illustrating the effect upon the peripheral vessels distal to sudden occlusion of the major artery by embolus or ligature right.

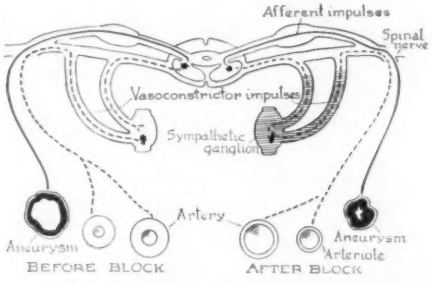

FIG. 7.—Illustrating the effect of sympathetic block upon aneurysms and the peripheral vessels.

sons[3] have shown experimentally that there is no flow of lymph. Cressman and Blalock[4] have made similar observations.

As previously stated, in the presence of vasospasm the circulation through the vasa vasorum may be so decreased that a definite interference with nutrition of the vessel wall may result in intra-arteriole thrombosis. Griffith and his coworkers[40] have recently demonstrated, experimentally, that the vasa vasorum of the femoral artery in the rat are definitely increased in number, as well as in diameter, following sympathectomy.

From a critical review of the effects of sympathectomy upon the vascular tree in the normal and obstructed major peripheral arteries, it can be definitely stated that sympathectomy counteracts the pathologic physiology produced by occlusion of major peripheral vessels (Table I) (Figs. 4, 6 and 7). Therefore, sympathectomy or sympathetic block (preferably the latter) as a therapeutic measure to prevent ischemic gangrene is definitely indicated in surgery of the major peripheral arteries. This method of developing the collateral circulation in surgery of the peripheral arteries was advocated by us[41] in December, 1933. In 1939, we[42, 43] again advocated this method as a preoperative therapeutic procedure to develop the collateral circulation and prevent the occurrence of ischemic gangrene in all surgical procedures upon the major peripheral arteries.

TABLE I

THE EFFECTS UPON THE PERIPHERAL VASCULAR TREE FOLLOWING OCCLUSION OF THE MAIN ARTERY	THE EFFECT OF SYMPATHECTOMY UPON THE PERIPHERAL VASCULAR TREE FOLLOWING OBSTRUCTION OF THE MAJOR PERIPHERAL ARTERY
(1) Spasm of main peripheral artery	(1) Vasodilation of main peripheral vessels
(2) Spasm of the collaterals	(2) Vasodilation of collaterals and increase in number
(3) Low arterial pressure distal to occlusion	(3) Return to normal of arterial pressure distal to occlusion
(4) Decreased peripheral venous pressure	(4) Return to normal of peripheral venous pressure
(5) Increased pressure proximal to occlusion	(5) Increased pressure proximal to occlusion
(6) Decreased blood volume flow per minute	(6) Sustained increased blood volume flow per minute through main artery and collaterals
(7) Decreased arteriolar pulsations	(7) Increased return of arteriolar pulsations
(8) Slowing and stasis of lymph flow	(8) Increased lymph flow
(9) Decreased flow through vasa vasorum	(9) Increased number and size of vasa vasorum
(10) Decrease in number of collaterals through which blood flows	(10) Increased number of collaterals
(11) Slow development of collaterals	(11) Rapid development of collaterals
(12) Degenerative changes in vessel wall	(12) Increased blood supply to vessel wall
(13) Occurrence of thrombosis	(13) Thrombosis extremely rare
(14) Muscle necrosis	(14) Increased blood supply to muscles
(15) Gangrene	(15) Ischemic gangrene prevented

Bird[10] and Plotkin[44] have also recommended the procedure in all cases of popliteal aneurysm as a preoperative therapeutic measure to prevent postoperative ischemic gangrene.

Leriche[23] has recently (1940) advocated sympathetic block in all cases of traumatic lesions of the major peripheral arteries in which ligation of the artery is necessary. In traumatic lesions of the peripheral arteries, we feel that in addition to the therapeutic methods advocated above, the intravenous injection of heparin (Murray and Best[45]) may be indicated to prevent secondary thrombosis resulting from trauma of the arterial wall. We do not advocate cervicodorsal or lumbar sympathectomy in such cases because we feel that the same results can be obtained by novocain and alcohol block of the sympathetic ganglia and chain.

Dos Santos[46] stated that following arterial occlusion for the cure of arterial and arteriovenous aneurysms he performs repeated sympathetic block postoperatively until a satisfactory and sustained collateral circulation has developed. DeBakey[47] stated that repeated novocain block of the sympathetics is preferable because a maximum response follows each block, whereas if the sympathetic ganglia and chain are destroyed either by surgical removal or alcoholic destruction, the maximum response occurs for a short period and cannot be repeated. Moreover, another disadvantage of alcohol is the possibility of producing an alcoholic neuritis of the somatic nerves. Alcoholic

neuritis occurs in about 30 per cent of the cases following block of the cervicodorsal sympathetics and in about 2 to 5 per cent of the lumbar injections.

Technic.—The technic of sympathetic block is not difficult and is practically without danger if only novocain or allied anesthetic drugs are used.

For the cervicodorsal sympathetic block we prefer the anterior approach of Leriche modified by DeBakey. By this method, using 10 cc. of 1 per cent novocain, the cervicodorsal ganglia are suffused with the anesthetic solution producing an extensive sympathetic block.

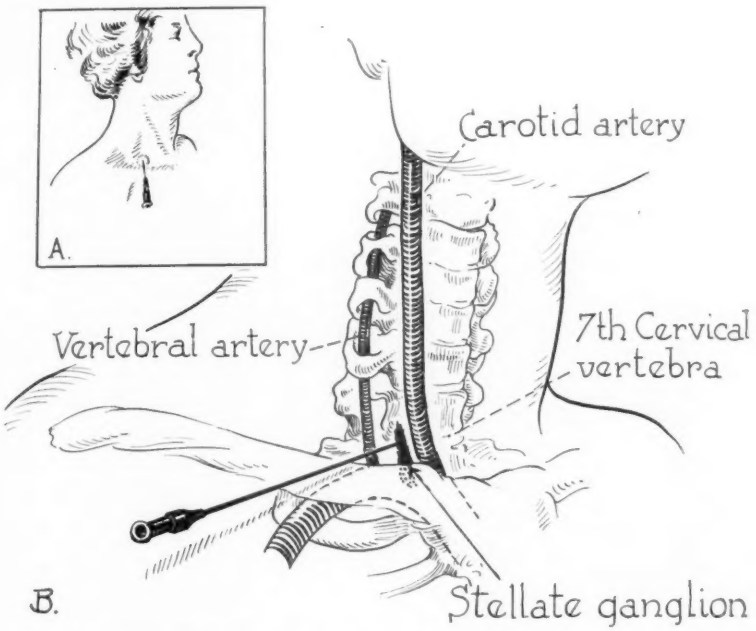

FIG. 8.—Drawing illustrating the technic for stellate ganglion injection. Insert shows anatomic landmarks used as a guide in the introduction of the needle.

The patient is placed in a supine position with the head turned slightly to the opposite side. A point is selected 1 cm. medial to the midpoint of the clavicle and immediately above its upper border (Fig. 8). The needle (spinal puncture needle) is then introduced inward and backward at an angle of 45° with the midline until it impinges against the anterolateral surface of the seventh cervical vertebra. The needle may impinge against the seventh cervical or against the ligament between the seventh cervical and first dorsal. If no blood is aspirated, 10 cc. of 1 or 2 per cent novocain is injected slowly. A successful block is indicated by the rapid appearance of a Horner's syndrome.

For lumbar sympathetic block, the patient can be placed either in the lateral position as shown in Figure 9 A or in the prone position with the lumbar vertebra and hips slightly elevated. The lumbar spinous processes are outlined by palpation. A point two fingers' breadth lateral to the upper border of the spinous process is located and a wheal of novocain injected intradermally to

mark the site of puncture. The first, second, third, and fourth lumbar transverse processes are so marked. A spinal puncture needle is now inserted vertically at each of the above cutaneous sites until the point impinges against the transverse process. The needle is then introduced for a distance of about 5 cm. (two and one-half fingers' breadth) (Fig. 9 C). Five cubic centimeters of novocain are now injected through each of four needles.

If it is decided to use alcohol, the needles are left *in situ* after injecting the novocain. The limb is observed for 20 to 30 minutes to determine whether

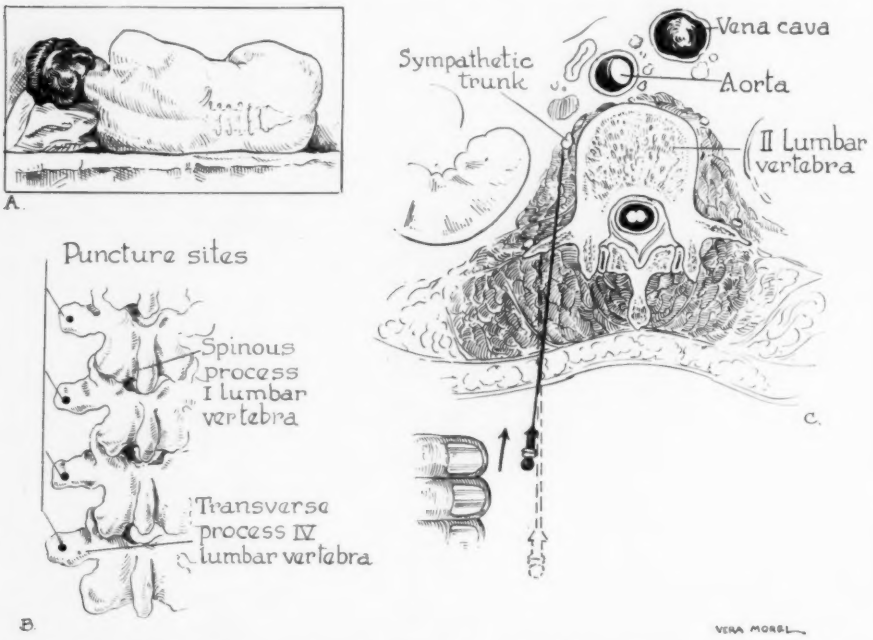

FIG. 9.—Drawing illustrating the technic of injecting the lumbar sympathetic ganglionated chain. A. Position of patient. B. Points of contact of the needle with the lumbar transverse process. C. The anatomic relations of surrounding structures to the needle.

the block has been successful. If successful, the surface temperature will be elevated and sweating will be inhibited. Five cubic centimeters of 95 per cent alcohol is then injected through each of the four needles. Before removing the needles a few drops of novocain are injected through them to wash out the alcohol.

Results.—We have employed the physiologic method, *i.e.,* sympathetic block, of increasing the collateral circulation as a preliminary procedure to the ligation of major peripheral arteries in ten cases. In all but two of these cases the collateral circulation was found to be inadequate by the Matas compressor test. In the other two cases, one of which was an aneurysm of the common iliac and the other a stab wound of the common femoral, the test could not be applied. Of these ten cases, one was a mycotic aneurysm of the right common iliac artery. Following sympathetic block and ligation of the common iliac

at its origin, there was no change in color and no decrease in temperature of the corresponding extremity. There were two cases of arterial aneurysm of the femoral artery and three cases of popliteal aneurysm which were cured by obliterative endo-aneurysmorrhaphy. Three of the cases consisted of arterio-venous aneurysm, two of which were femoral and one was popliteal. These were treated by quadruple ligation. There was one case of stab wound of the common femoral which required ligation. In none of these cases of ligation of the major peripheral arteries treated by preliminary sympathetic block was there any evidence of ischemia or deficiency of the peripheral circulation.

We have also used sympathetic block in four cases of embolus of the femoral artery. In one case the embolus was removed after sympathetic block. The other three cases were not operated upon. In all these cases the classic clinical manifestations of arterial embolism were present. Following novocain block of the lumbar sympathetic ganglia and chain on the affected side, there was a loss of numbness and a return to normal of color and temperature of the extremity. In peripheral arterial embolism there is not only a high incidence of ischemic gangrene but a high mortality, the gangrene increasing the mortality. Therefore, we believe that sympathetic block will not only materially decrease the incidence of ischemic gangrene but will also lower the immediate mortality.

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DISCUSSION.—RUDOLPH MATAS (New Orleans, La.): The excellence of Doctor Gage's presentation suggests a contrast between the past and present methods of introducing papers at these meetings which are no doubt best appreciated by the older members, who, like the speaker, have lived to enjoy the modern outlook, so strikingly exhibited by Doctor Gage. With the marvelous aid of contemporary cinematography and short, crisp, tabloid, lantern-slide condensations, a long dissertation is abridged with enormous economy of words without sacrifice of lucidity or precision. Formerly, we spoke of a bird's-eye view of a scene or subject. Now, we survey an encyclopedic panorama with all the speed, sweep and effectiveness of an airplane view. In this way, our surgical programs are being made increasingly attractive and instructive by their pictorial and epitomized visualizations.

. . . . . .

The salient feature of Doctors Gage and Ochsner's thesis is their advocacy of alcoholic injections ("chemical section") of the regional neuroganglionic sympathetic, as a preventive of ischemic gangrene in all operations that may necessitate the occlusion, excision or obliteration of the major peripheral arteries. Their experience, well-backed by others, has given them confidence in the value of the vasodilator effect of neuroganglionic alcoholization as an effective means of accelerating and dilating the collaterals when the circulation in the main artery is suddenly blocked by a ligature for arterial wounds, for aneurysm, or, when angiospasm, an embolus or a thrombus blocks the circulation.

Based upon their experience, the authors advocate the pre- and postoperative practice of chemical ganglionectomy by alcoholic injections as a systematic, or routine procedure, in all occlusive operations upon the main arteries of the upper and lower extremities.

When ischemic gangrene follows the ligation, or other obstacle to the circulation, in the main artery of a region or extremity, the fatal ischemia is attributed to a failure of the collaterals to carry on the circulation beyond the ligation or obstacle. The causes of this failure of the collaterals are numerous, including the classic pathogeny, namely, congenital anatomic defects; chronic and acute arterial disease; relative viability of the tissues or organs involved in the ischemic area; stasis from venous obstruction; profound anemia and the

cardiocirculatory failure of hemorrhage and shock, with vasoconstriction or angiospasm as a contributing factor, *etc.*

Since the benefit of sympathetic neuroganglionic alcoholization is dependent upon vasomotor paresis, with secondary dilatation of the arteries in the vascular bed, an activation of the collateral circulation is expected to follow the vasoparetic overflow. While the vasodilating effect of the alcoholic injections would indicate this procedure as especially effective in the rare cases in which angiospasm is a dominant factor, it is logical to believe that the vasodilating effect of the injection would also benefit the local anemias that are still responsive to sympathetic influences. Unfortunately, the patients who would be most benefited by vasodilation are, in the majority, victims of chronic or acute vascular disease—the senile and presenile arteriosclerotics, the thromboangiitic, and others who are largely irresponsive to vasomotor control.

The importance of the relative viability of the tissues or organs supplied by the ligated or obstructed artery in determining the necrogenic effect of an acute ischemia is well exemplified in the coma and brain disorders that follow carotid ligations, and in the necrotizing effect of embolic obstructions in the pulmonary, the mesenteric and other splanchnic infarctions. Again, quite apart from arterial disease as a cause of ischemic gangrene are the ligations of the great vessels in young soldiers who are picked up from the battle field exsanguinated, shocked, after days of long exposure to cold and nights of sleepless vigilance and terrifying experiences. Under such conditions the ligation of the main artery of a limb is almost sure to end in gangrene, and in coma and death, if the cartoid is ligated. In such cases, any attempt to prevent an ischemic gangrene by sympathetic ganglionectomy would prove not only an illusion, but a therapeutic parody. The experience of military surgeons in modern warfare—especially since blood transfusion has come into vogue, as in the late civil war in Spain, in which "blood banks" and "canned blood" were more available than in previous wars—has demonstrated that the surest preventive of ischemic gangrene following the ligation of the great arteries, including the carotids, is blood transfusion, copiously and repeatedly administered, until a living and actively functioning cardiocirculatory balance is restored.

In considering the adoption of sympathetic ganglionic alcoholization as a routine procedure, we should remember the variations in the distribution of the collateral branches of the main arteries which in the upper extremity (subclavio-axillary tract) are so free and abundant that ischemic gangrene after the ligation of the subclavian and the arteries of the arm may be regarded as a negligible risk outside of the shocked and exsanguinated wounded previously referred to. I know, personally, of 49 ligations and band occlusions of the subclavian artery for aneurysm with only one partial gangrene of the hand.

It is only as a sequela to infected emboli or propagated thrombi that gangrene is to be feared in the subclavian tract.

We realize the seemingly improvident paucity of collaterals in the lower extremities as compared with the abundance in the upper extremity when extirpating tumors or obliterating aneurysms of the terminal popliteal which involve its tibioperoneal trifurcation. It is in dealing with the arterial wounds, lesions (aneurysms) and other obstructive circulatory disorders of the lower extremities that lumbar sympathectomy by alcoholization finds its most favorable field of application, though, even here, as the patients advance in years, its value in the prophylaxis of surgical ischemia must be regarded only in the light of an adjunct or auxiliary function.

Despite the limitations of chemical ganglionectomy by alcoholization as a preventive of ischemic gangrene, it is a valuable addition to the resource of

vascular surgery in dealing with the special indications of the method. The experience of the authors, especially Doctor Gage, in aneurysm, fully attests its value in promoting the collateral circulation when there is reason to doubt its efficiency before operation. Despite my great interest in the prophylaxis of ischemic gangrene in the surgery of aneurysm, I have been able to do without sympathetic ganglionectomy by alcohol or otherwise, in my practice, but I have no doubt that the alcoholization of the lumbar ganglia would have hastened the development of the collaterals in some cases. Though fairly safe in the expert hands of the authors, the alcoholization of the lumbar ganglia by the paravertebral route cannot be regarded as an innocuous procedure. My long experience in paravertebral splanchnic anesthesia with novocain solution, when the Kappis method was in vogue, has made it plain to me how obstinate neuritic pains and paresthesiae in the distribution of the spinal nerves may result from the diffusion of the alcohol in the paravertebral spaces.

Again, in considering alcoholic sympathectomy as a routine procedure for the prophylaxis of ischemic gangrene, we may say that since the methods of testing the efficiency of the collateral circulation in the neck and extremities have become fairly dependable and generally available, we are able to determine beforehand, with close approximation, what the behavior of the limb will be after ligation of its main artery. If there is clear evidence that the collateral circulation is efficient, it would seem unnecessary to resort to a prophylactic lumbar sympathectomy by alcoholization; nor need we worry in dealing with an aneurysm in our choice between a radical and a conservative operation.

If there is proof that the temporary occlusion of the main artery with our mechanical compressor* applied precisely at the prospective site of the ligation is followed by prolonged pallor, indicative of an inefficient collateral circulation, we have time, at least in the majority of cases of aneurysm, to train and develop the collaterals by the method of direct intermittent mechanical compression which I introduced 38 years ago and have continued to use effectively ever since. This procedure, which Doctor Gage has utilized and described with great fidelity, is usually combined with other classic methods of developing the peripheral circulation, such as contrast baths, diathermy, intermittent negative and positive pressure exercises (Pavaex, Collens-Wilensky, de Takats, *et al.*). When there is no hurry, the slow and simple methods of collateral training will do; but in acute, accidental injuries when ligations are required for the control of hemorrhage, and there is little opportunity for vascular exercises, the method of chemical lumbar ganglionectomy with alcohol will probably find, here, one of its most useful applications, as an adjunct to transfusion and other cardiovascular restoratives.

Dr. Idys Mims Gage (New Orleans, La.): I not only wish to thank Doctor Matas for his kind discussion of our paper, but also to express my gratitude and deep appreciation to him for his many kindnesses, because his tutelage and constant encouragement have been of inestimable value to me.

We did not offer this procedure of sympathetic block as a "cure-all" but recommend it as another method for the developing and maintaining of adequate collateral circulation in the presence of sudden occlusion of a main peripheral artery. The procedure has given such excellent results in our hands that we wish to suggest its use to those interested in vascular surgery.

* Matas, Rudolph: Annals of Surgery, **53**, 1–43, January, 1911. *Idem:* Amer. Jour. Surg., N.S. **24**, 692–698, June, 1934.

CIRCULATORY DISTURBANCES PRODUCED BY EXTENSIVE ANGIOMATA OF THE LOWER EXTREMITIES ASSOCIATED WITH VARICOSE VEINS*

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THE SIMPLE ANGIOMA is of significance chiefly because of its disfiguring appearance unless it happens to be situated in some vital organ. When it is freely connected with the general circulation, however, so that the pressure within the smaller vessels is raised, it may increase greatly in size. Under such circumstances, a lesion which was originally simply a "birth mark," unsightly but harmless, may alter its characteristics so as to give rise to serious symptoms. Not only may the circulation locally be impaired and lead to ulceration and gangrene, but the effects on the systemic circulation may be harmful.

Free connections between the angioma and the arterial side of the circulation lead to the formation of a cirsoid aneurysm. The physiologic disturbances which result from this condition have been fully described by Reid[1] and Holman.[2] When the angioma is in the lower extremity and is freely connected with the venous side through veins with defective valves, it is subjected to increased pressure. Dilatation of the smaller vessels takes place and gives rise to symptoms, both local and general, which, though less spectacular than those accompanying the cirsoid aneurysm, may be serious and incapacitating.

The symptom complex of extensive angiomata of the lower extremity with varicose veins was first described by Devouges,[3] in 1856. This condition was found to be associated with osteohypertrophy and attention was directed chiefly to this aspect of the patient's picture. In 1869, Trelat and Monod[4] reviewed the literature on osteohypertrophy and described the associated vascular lesions. Klippel and Trenaunay[5] originated the term "*naevus variqueux osteo-hypertrophique*," in 1900. They gathered reports of 14 patients from the literature and described an additional case. Three variations of the symptom complex were encountered: Those with the angioma alone; those in which the osteohypertrophy was the outstanding feature; and, finally, those with associated varicose veins. In their experience, osteohypertrophy was frequently encountered. It was unusual not to find varicose veins. The varices were rarely seen in infancy but the patients developed varicosities at an early age. Varicose ulcers often developed. The peripheral pulses were normal but the skin temperature was higher on the affected side. Numerous reports of cases have since appeared in the French literature—Van Neck,[6] in 1925; Pautrier and Ullmo,[7] in 1928; Sorrel,[8] in 1932; Gougerot and Lortat-Jacob,[9]

* Read before the American Surgical Association, St. Louis, Mo., May 1, 2, 3, 1940.

in 1934; Alajouanine and Thurel,[10] in 1935; Radulesco,[11] in 1935; Pautrier and Lang,[12] in 1937; and Touraine, Duperrat and Baudouin,[13] in 1938. In this country, cases have been reported by Homans,[14] in 1922; Reid,[1] in 1925; Kleinberg,[15] in 1930; DeTakats,[16] in 1932; and Wise and Lisansky,[17] in 1938.

During the past year, we have observed three patients with extensive angiomata of the lower extremities which were in direct communication with the general circulation through veins with defective valves. The circulatory disturbances which these patients presented form the subject of the present communication.

CASE REPORTS*

Case 1.—Hospital of University of Pennsylvania, No. 40306: B. L., white, female, age 17, was admitted to the Surgical Service of Dr. I. S. Ravdin, with the complaint of a "sore and swollen" right foot and attacks of syncope on standing. The patient had a large angioma of the right leg extending from the crest of the ilium to the sole of the foot, which had given rise to no subjective symptoms; a large vein on the medial aspect of the thigh had, however, been injected at the age of 11. At the age of 12, shortening of this leg was noticed. Four months before admission she developed phlebitis of the right leg. After recovery from the acute attack, it was first noticed that the angioma became turgid and suffused when the patient was in the erect posture, and that she frequently fainted when standing.

Physical Examination.—The patient was found to have varicose veins of the right leg which filled from the saphenous and from a communicating vein of the lower thigh. The angioma filled rapidly through these veins but the reflux could be prevented by the application of a venous tourniquet. On standing, as shown in the first section of Chart 1, the blood pressure fell from 135/85 to 90/70, and the pulse rate increased from 60 to 152. Upon release of the tourniquet, which had completely occluded the circulation above the knee, the volume of the right leg, measured by displacement of water, increased by 200 cc. within the first minute, as shown in the top curve of Chart 2. On the left (normal) side, the increase was only 25 cc., as shown in the bottom curve of Chart 2. Specimens of blood were obtained simultaneously from the veins of both lower legs and from both femoral veins, and the color of the blood obtained from all the veins was the same. At operation, the right long saphenous vein was ligated at its junction with the femoral together with four large branches, each the size of a normal femoral vein.

After operation, the rate of increase in leg volume after release of the tourniquet was found to be reduced, as shown in the second curve of Chart 2. The blood pressure and pulse changes on standing were not as marked although, as the second section of Chart 1 illustrates, she still fainted in the erect posture. The angioma of the thigh and hip did not become suffused on standing but the veins of the lower leg still filled from above. She was followed for a year, during which time the tissues of the lower leg were supported with an elastic stocking. She was examined a year later and at this time she no longer fainted on standing. The third section of Chart 1 illustrates the changes in blood pressure and pulse when she stood up. Since the angioma of the lower leg still filled from above, the saphenous vein was ligated at the point where the communicating vein from the femoral emptied into it. Three months later, the increase in the volume of the lower leg after release of a tourniquet was only 90 cc., as shown in the third curve of Chart 2, and the blood pressure and pulse changes on standing were almost normal. As the fourth section of Chart 1 shows, there was no fall in systolic pressure, and only

*Within the past three months, all three of the patients described in this article have been operated upon for further ligation of communicating veins. In each case when the dissection has been carried out through the superficial angioma, difficulty in wound healing was encountered, apparently due to persistent oozing of blood from minute vessels. Healing was delayed and the period of hospitalization increased.

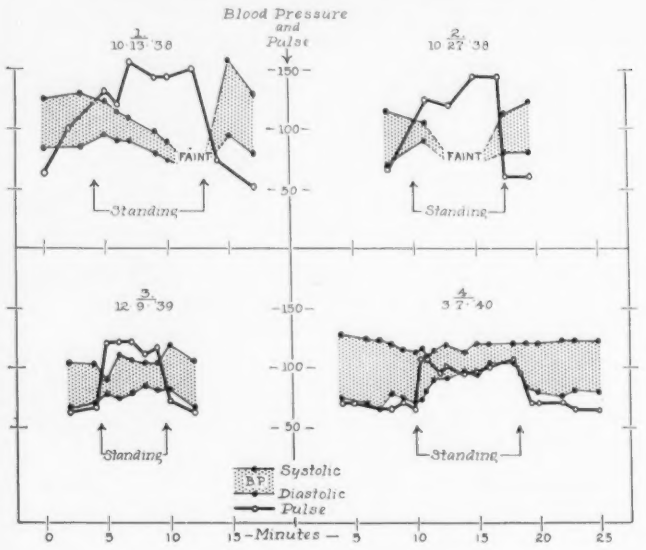

CHART 1.—Effect of standing on blood pressure and pulse rate of patient B. L.: (1) Before ligation of long saphenous on right; (2 and 3) after ligation of long saphenous on right; (4) after ligation of communicating vein of lower thigh.

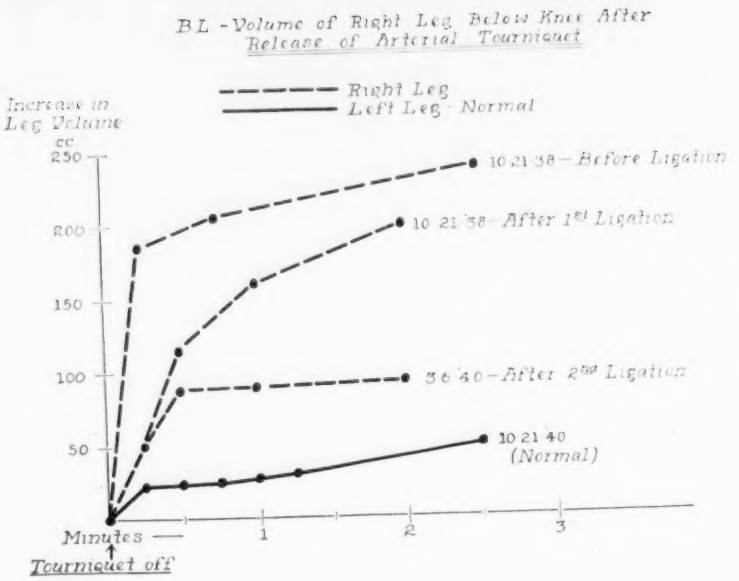

CHART 2.—Increase in volume of right lower leg of patient B. L. after release of tourniquet applied above the knee; before and after ligation of the saphenous vein; and after ligation of communicating vein in the lower thigh. The increase in volume of the left normal leg under similar circumstances is shown in the bottom curve.

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a moderate rise in pulse rate. She was free from symptoms, and the angioma had decreased in size so that it was no more noticeable than it had been before her attack of phlebitis.

Case 2.—Pennsylvania Hospital, No. 36544: A. E., white, male, age 23, complained chiefly of pain and repeated attacks of phlebitis in varicosities of the right leg. At birth, he was found to have multiple angiomata of the right lower extremity, penis and right half of the scrotum. There was a general osteohypertrophy of both feet but particularly of the middle three toes of the right foot and the second and third toes of the left foot. The appearance of the feet is shown in Figure 1. Varicose veins of the right leg, first noticed at the age of 12, had been subject to recurrent attacks of thrombophlebitis with such severe pain that he developed a 60° flexion contracture of the right knee. During the period 1934–1937, he was operated upon four times for excision of thrombosed and varicosed veins and correction of the deformity of the knee. On examination, it was found that the varicose veins of the right leg were in free communication with large blood sinuses on the posterolateral aspect of the thigh and leg. Figure 2 shows the location of these "lakes." Their filling could be prevented by the application of a venous tourniquet to the upper thigh. The long saphenous vein was not involved, and the point of incompetence appeared to be in the region of the gluteal veins. With the patient lying "head down," 30 per cent diodrast was injected into the top of this venous column and a roentgenogram taken. Figure 3 illustrates how the dilated vein was seen to pass through the fascia at the lower border of the gluteus maximus muscle and run up through the sciatic notch. An additional portal of entry appeared to be in the region of the superior gluteal vein. The varices of the upper portion of the buttocks also filled.

At operation, two large veins were divided at the inferior margin of the gluteus maximus. The tissue was extremely vascular and the bleeding was controlled with difficulty. After operation, the varices still filled from above but the filling was slower. Before operation, the pulse rate increased from 60 to 124 when he stood up. After operation, the pulse rate increased only to 104. Although the systolic blood pressure did not fall, there was an increase in the diastolic blood pressure in the erect posture. Further localization of the point of incompetent valves of the communicating veins is to be sought.

Case 3.—Graduate Hospital of the University of Pennsylvania, No. 142803: D. O., white, male, age 19, complained chiefly of edema of the legs and ankles. At birth, it was noticed that he had multiple vascular and other congenital anomalies. There was hypertrophy of the bones of both feet and the left upper extremity. Large angiomata were present over both lower extremities, penis and scrotum. The angioma on the left side extended from the level of the umbilicus and the crest of the ilium to the sole of the feet. The index and ring fingers of the left hand were fused. At the age of 12, he developed varicose veins of both legs. The left breast hypertrophied, and there was excessive growth of hair on this breast.

Physical Examination.—The varices of the left leg were found to fill, largely, from the long saphenous system. On the right side, there was free communication between the angioma and the short saphenous system but filling on both sides could be prevented by the application of venous tourniquets. The appearance of the feet and legs is shown in Figure 4. The angiomata became suffused with blood when the patient stood up, while the blood pressure fell from 120/70 to 80/60, and the pulse rate increased from 65 to 144, as shown in the first section of Chart 3. The volume of the left lower leg increased by 475 cc. within one minute upon release of a tourniquet which had been applied above the knee. The oxygen saturations of venous blood, taken simultaneously from one of the veins of the leg and from the antecubital vein, were 67 and 69 per cent, respectively.

At operation, the left long, and the right short, saphenous veins were ligated at the points where they pierced the deep fascia. After operation, the filling of the angiomata was delayed and the blood pressure no longer fell on standing, as shown in the second section of Chart 3. Further examination revealed additional points of incompetency

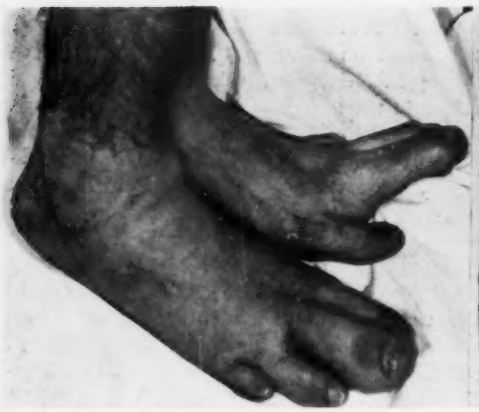

FIG. 1.

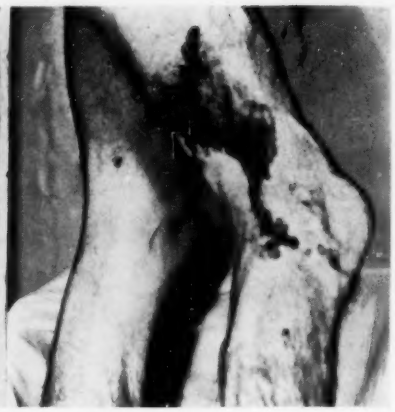

FIG. 2.

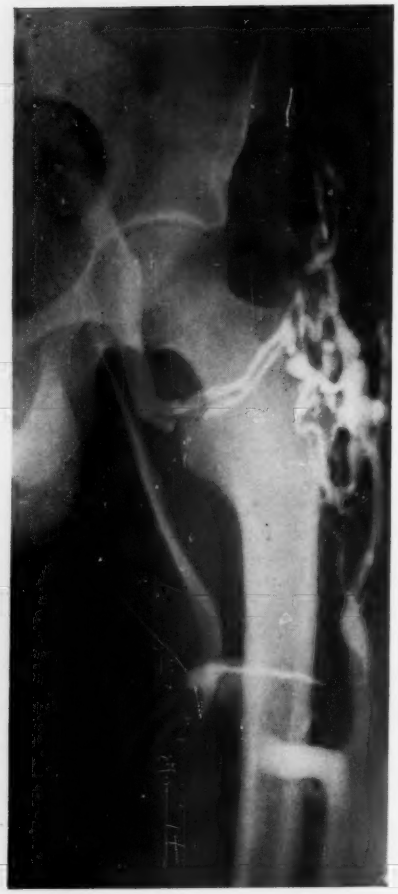

FIG. 3.

FIG. 1.—Appearance of the feet in patient A. E., 10 years after Doctor Gill had excised the epiphyseal cartilages of the metatarsal bones of both feet.

FIG. 2.—Extensive angiomata and varicose veins on the lateral aspect of the thigh and leg in patient A. E.

FIG. 3.—Visualization of the veins of patient A. E. after injection of 30 cc. of 30 per cent diodrast into the venous "lake" on the lateral aspect of the thigh.

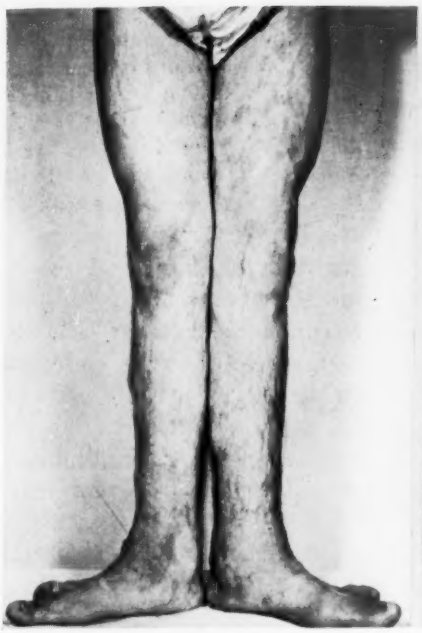

FIG. 4.—Appearance of the legs and feet of patient D. O. before ligation of veins.

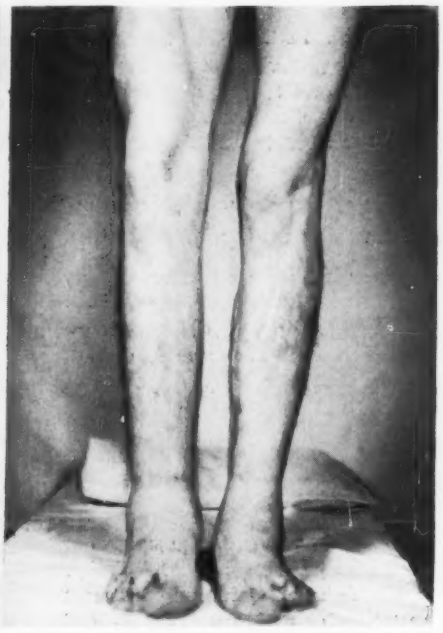

FIG. 5.—Appearance of the legs of patient D. O. after 30 seconds of standing, after right short, and left long saphenous ligation.

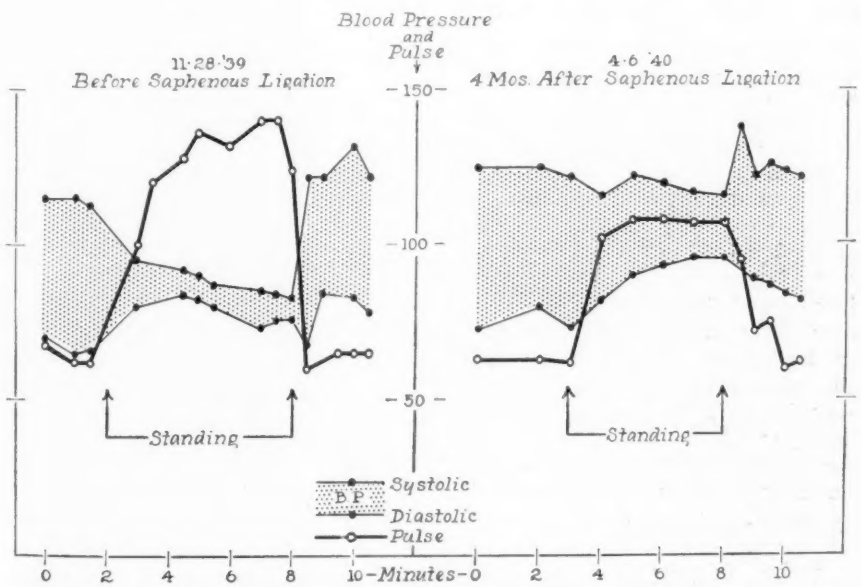

CHART 3.—Effect of standing on blood pressure and pulse rate of patient D. O. before, and four months after, vein ligations.

of the valves between the deep and superficial systems, in the region of the inferior gluteal on the right and in the midthigh on the left side. The filling was slow, as the photograph taken after 30 seconds of standing after operation indicates, as shown in Figure 5. Ligation of these veins is now planned.

Discussion.—The essential difference between the condition which these patients presented and the cirsoid aneurysm is the source of the filling. In the latter condition, there are abnormal connections of the angioma with the arterial side of the circulation. In the angioma of the lower extremity associated with varicose veins, the filling is from the venous side. It is only in the lower extremity that the venous pressure, in the absence of competent valves, is sufficiently high to produce expansion of the vascular bed. No case of angioma with varicose veins of the upper extremity has been observed. The fact that filling occurred from the venous rather than from the arterial side was indicated by the fact that there was no pulsation of the veins, and that the color and oxygen saturation of the venous blood taken from the affected extremities in two of our cases was identical with that taken from unaffected regions. An additional point of differentiation was the fact that filling could be prevented by the application of a venous tourniquet. If the angiomata had filled from the arterial side, the veins would have increased in size when a tourniquet obstructed the venous return. Finally, the reduction in size after ligation of the veins which communicated with the angiomata served as additional evidence of their venous character.

The presence of osteohypertrophy suggested a similarity between the angioma with varicose veins and the cirsoid aneurysm since overgrowth of bone is constantly noted in the latter condition. The hypertrophy which was observed in the second and third cases, however, was spotty since all of the bones of the extremity were not involved. Again, in the patient who had only the angioma until she developed acute phlebitis, there was shortening of the affected extremity, similar to that described in Reid's[18] patient.

The local symptoms in the first and third cases seemed to be due to the varicose condition, *i.e.*, swelling and discomfort. Klippel and Trenaunay[5] comment upon the frequency with which varicose ulcers appeared in the cases which they reviewed. The outstanding symptom in our second case was pain. It is probably significant that this patient had repeated attacks of phlebitis in the superficial veins of the leg. Similar cases of pain in this condition were reported by Sorrel,[8] in 1932, and Pautrier and Lang,[12] in 1937.

Reid[19] was the first to call attention to the effects of the venous reflux on the systemic circulation. In his patient, the angioma was apparently not complicated by the presence of varicose veins. The blood pressure fell from 130/85 to 126/84, and the pulse rate increased from 86 to 116 on standing, but after ten minutes the pulse rate fell to 104. These figures are comparable to those obtained in all our patients after the main channels of reflux had been closed. The volume of blood lost from the general circulation into the angiomata was large. In the first patient, the increase in volume of the lower leg upon release of a tourniquet was 250 cc., and yet the more extensive part

of the angioma over the thigh and hip was above this region. In the third patient, the increase in volume of the left lower leg was 475 cc. The angioma extended up to the middle of the trunk on this side and the other leg was also involved. The increase in pulse rate, rise in diastolic and fall in systolic pressure may be compared to the physiologic effects of hemorrhage. As the return of blood to the right side of the heart diminished, there was a compensatory increase in pulse rate with vasoconstriction. The pulse pressure decreased both by a decrease in systolic blood pressure and a rise in diastolic pressure. Compensation for the impaired cardiac return was accomplished by these protective mechanisms. When the loss of circulating blood volume was too great, the cerebral tissues become anoxic and fainting occurred.

The venous valve is probably of major significance in the development of symptoms. In the two male patients certain valves which protected the superficial system from excessive pressure were absent or defective presumably on a congenital basis. The fact that the first patient suffered no ill effects until the valves had been damaged by phlebitis is in accord with this concept, since it has been shown by Edwards and Edwards[20] that the venous valve is destroyed during the process of recanalization after the thrombosis.

The usual treatment of this condition has been excision (Pautrier and Lang,[12] Kleinberg[15] and DeTakats[16]), while injections were used by Light.[21] Roentgenotherapy was considered useless for the superficial diffuse angiomata by Taylor.[22] Homans[23] obtained permanent relief in his case by excision of the contributing vein at its head, and injections of sclerosing solutions into the peripheral portion. Reid[18] stated: "In my experience an extensive angioma involving the entire leg was cured, by thrombosis, after the excision of a very small portion of it." Since defective valves appear to be, etiologically, significant, rational treatment would appear to be ligation of the incompetent vein with subsequent sclerosis of the remaining segment if necessary. The progressive decrease in the capacity of the angiomata after the head of pressure was reduced in our cases, by ligation of the vein with the defective valve, may indicate that subsequent injections of sclerosing solutions will not be necessary.

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HEPARIN IN THE PREVENTION OF PERITONEAL ADHESIONS*

REPORT OF PROGRESS

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A PRELIMINARY REPORT,[1] recently presented, suggested that heparin, intra-abdominally administered, is effective in preventing the formation and reformation of peritoneal adhesions in the rabbit and dog. Ten dogs, receiving 3,000 units of heparin in solution in single daily administrations at operation and on each of two postoperative days, presented an adhesion reformation rate of 26 per cent, whereas the corresponding rate was 157 per cent in the control group of 20 animals, in which either no solution, normal saline solution, or amniotic fluid concentrate was administered intraperitoneally. In other words, after heparin the count of adhesions was about one-fourth of the count at the time of injection of the substance; in the control group the adhesion count showed more than half as many again at the final stage as compared to the number of adhesions divided.† Some doubts were expressed in regard to the safety of intraperitoneal heparinization from the point of view of hemorrhage on the basis of three fatal hemorrhages in 24 dogs. This high incidence of bleeding was believed to be the result of inadequate hemostasis at the time of dividing the adhesions.

The present report of progress offers evidence on three phases of the general study: (1) Intraperitoneal dosage; (2) the danger of hemorrhage; (3) the effect of intraperitoneal heparin in the freshly contaminated abdomen.

METHODS EMPLOYED.—Dogs were employed throughout. The method for creating peritoneal adhesions was that used in the previous investigation.[1] Adhesions were consistently produced by perforating the appendix and smearing a small, measured quantity of its expressed contents over the terminal ileum and the adjacent cecum. In recent experiments, light, dry gauze scarification of the terminal ileum was an added technic preliminary to contamination. This procedure was found to localize the adhesions more successfully. The appendiceal perforations were not closed. Six weeks later the resulting

* Read before the American Surgical Association, St. Louis, Mo., May 1, 2, 3, 1940.

† Since publishing the original paper, five additional control animals were studied similarly with papain made up in Hartman's solution in a concentration of 1-20,000, as recommended by Ochsner.[2] The animals treated with papain presented a reformation of adhesions of 117 per cent. The results with papain were numerically the best of the control studies, bringing the average rate of adhesion reformation down to 147 per cent. The papain employed was obtained through the courtesy of Parke, Davis and Co., Detroit, Mich.

adhesions were divided and heparin solution in varying doses was administered intraperitoneally. In some animals (Table 1) additional heparin was given by paracentesis on one or more subsequent days. A third operation was performed two weeks later, at which time the number of reformed adhesions was observed and recorded.* Powdered heparin was purchased from the Connaught Laboratories, at the University of Toronto, and was dissolved in sterile water at the time of use. Normal saline was originally employed as the vehicle but was discarded when the reformation results with saline solution alone were found to be the highest in the control series. A number of animals in these groups have been discarded on account of wound infections communicating with the peritoneal cavity as the result of an epidemic of contamination of the cages.

In the experiments dealing with the contaminated peritoneum, the heparin solution (3,000 units) was first introduced at the time of perforating the appendix rather than after adhesions had formed. Two additional doses were given by paracentesis on successive postoperative days.

RESULTS.—*Dosage Experiments:* Table I presents the results of the dosage experiments in which both the number of single daily administrations and the quantity of heparin per dose were varied. The figures indicate that more than two intraperitoneal doses are necessary, even when a relatively large number of units of heparin are administered, and that more than three doses do not improve the results. The number of reformed adhesions following two daily doses of 1,000 units each is appreciably greater than when 3,000 units are employed. When three administrations are made, the results of the 1,000 unit dose and the 3,000 unit dose are approximately equal.

TABLE I

REFORMATION OF ADHESIONS

Results Two Weeks Following Separation of Previously Produced Adhesions in the Dog After the Introduction of Heparin Solution Intraperitoneally by Paracentesis

Number of Dogs	Number of Daily Heparin Injections	Units of Heparin Given per Injection	Average Number of Adhesions Separated	Average Number of Adhesions Reformed	Average Per Cent of Adhesions Reformed
10	1	3,000	6.3	5.8	92
5	1	9,000	15.6	9.6	62
5	2	1,000	15.4	13.8	89
10	2	3,000	16.0	8.4	52
10	3	1,000	8.4	2.4	29
10	3	3,000	9.7	2.6	26
6	4	3,000	15.8	5.3	33

Hemorrhage.—In experiments to date with intraperitoneal heparin administration, no further intra-abdominal hemorrhages have occurred. There

* The attempt at a quantitative method of recording results is, of course, inaccurate. It offers, however, the only objective method available short of direct observation of the animals.

have been to date 75 consecutive introductions of heparin without hemorrhage, and only three hemorrhages in a present total of 101 dogs.

Heparin in the Contaminated Peritoneum.—Table II presents the results of the introduction of heparin into the freshly contaminated peritoneum in 17 dogs as compared with the results in a group of 100 consecutive control animals in which the peritoneum was contaminated for the creation of adhesions for later study. Whereas 43 per cent of the control animals died of peritonitis, 53 per cent of the heparin dogs succumbed to this complication. In the animals that survived, however, the average number of adhesions formed in the heparin dogs was less than one-third of that formed in the controls.

TABLE II

HEPARIN AND CONTAMINATION

Results Following the Intraperitoneal Introduction of Heparin at the Time of Perforation of the Appendix

Group	Number of Dogs	Mortality Rate	Average Number of Adhesions in Living Dogs
No solution	100	43%	9.4
Heparin 3,000 units at operation and two additional doses	17	53%	2.9

DISCUSSION.—The absolute figures furnished by the present experiments confirm the earlier conclusion that heparinization of the peritoneal exudate in dogs inhibits the reformation of divided intra-abdominal adhesions. The suggestion of a curve dependent upon dosage, as presented in Table I, adds further evidence of a definite heparin effect. To this may also be added the low numerical incidence of adhesions in the surviving dogs, presented in Table II.

The study of dosage is as yet incomplete. The method of attack is necessarily slow and final conclusions may be delayed. Furthermore, for reasons to be discussed below, the ultimate determination of the optimum dosage of heparin given intraperitoneally may not be pertinent. Certainly, for the moment, it is clear that more than two intraperitoneal doses are necessary, and that four daily doses do not present improved results over those following three daily doses. The minimum effective dose per day for three administrations has not yet been determined.

The freedom from further occurrence of hemorrhage has paralleled greater operative attention to hemostasis. It is probable, therefore, that the earlier explanation of the cause of the trouble at first encountered, namely, inaccurate hemostasis, is correct. We now feel that hemorrhage is not a danger, if bleeding can be completely controlled before introduction of the heparin solution.

In the earlier experiments with rabbits,[1] it was surprising that 100 per cent

of recoveries occurred when heparin was introduced into the peritoneum at the time of perforation of the appendix. It might have been expected that all such animals would die of general peritonitis on account of the assumed absence of fibrinous adhesions. In the present group of dog experiments, almost equally surprising results occurred. It had been supposed that at least 90 per cent of the dogs in which heparin had been introduced at the time of perforation would die of peritonitis. In contrast to this expectation, only 53 per cent died, a mortality not strikingly different from that among the controls. This seems to indicate that heparin may not materially increase the danger of peritonitis when the peritoneum is soiled. We are not yet ready to suggest a modification of the traditionally accepted ideas of peritoneal defense against infection as at least partially effected through fibrinous adhesion about the source of contamination. We feel, however, that these results indicate the possible usefulness of heparin in the contaminated peritoneum provided the source of contamination is not still present, as for instance following intestinal suture. Further experiments in this connection are contemplated.

The studies so far prosecuted do not yet permit of clinical application. We are convinced of the effectiveness of heparin and of its safety. The method of administration and the optimum dosage are still undetermined. It must be pointed out that repeated daily intraperitoneal administration is not well adapted to clinical use. Postoperative paracentesis in patients may be uncomfortable and may present danger. Three other methods of administration are now under investigation: (1) The first is based on the possibility that an intraperitoneal exudate may be uncoagulable if the blood plasma from which it is derived is first rendered relatively uncoagulable by subcutaneous or intravenous heparinization. In these studies heparinization of the animal is begun by these routes as soon as the operation for division of adhesions is completed. (2) The second method combines intraperitoneal administration of heparin at the time of division of adhesions (as in the present experiments) with subcutaneous or intravenous heparinization in an attempt to prolong the local effect. (3) The third attack is a study of intraperitoneal drip heparin administration through an inlying rubber tube introduced at operation.

Until the best method has been worked out in the dog application to the clinical case must be delayed. As stated in the earlier report the possible success of the method in clinical surgery must, at least for a long period, be judged on laboratory rather than clinical evidence.

CONCLUSIONS

(1) Intraperitoneal heparinization in three daily doses of at least 1,000 units largely prevents the reformation of divided peritoneal adhesions in the dog.

(2) Hemorrhage following the intraperitoneal administration of heparin is not a danger if hemostasis is complete before the heparin is administered.

(3) Contamination of the peritoneum does not appear to be a contraindication to the intraperitoneal use of heparin.

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DISCUSSION.—DR. HARVEY B. STONE (Baltimore, Md.): Every general surgeon, I think, must be greatly interested in any proposed method of diminishing abdominal adhesions, and I personally have been particularly interested in this because, many years ago, one of the earliest pieces of experimental work that I undertook concerned itself with measures designed to prevent adhesions—and I might say in passing, it was completely unsuccessful. So when Doctor Lehman's work came to my attention, naturally, I was immensely interested in it, because it seemed to me that it was the most logical and hopeful attack on the problem that I had personally ever heard of.

The purpose of my speaking at all now is to report very briefly some uncompleted work which is being carried on by Doctors Owings and Hewitt, two of the younger men connected with the Hopkins Clinic, which they have given me permission to speak about. They are working on the same issue, namely, heparin as a preventive of abdominal adhesions, but have purposely modified, in several directions, the technic employed by Doctor Lehman, in order to explore varieties of procedure which may increase the success of the method. They have used various means of inducing adhesions, and the one, at the moment, which they have found most effective and, in their judgment at least, most suitable for comparative studies, is the production of a chemical irritation by painting of serous surfaces with small amounts of ferric chloride. Their experimental animals have been cats instead of either dogs or rabbits. They have used a different preparation of heparin, or at least heparin from a different source from that employed by Doctor Lehman, so that from many aspects of detail, their series will give a comparable study to his. Their work is by no means completed; in fact, I am requested to say that they are not in a position to express any opinion as yet about the results but merely to say what they have done.

First, I might say they have had no hemorrhages at all in their series of animals as a result of the introduction of heparin. They have found, beginning with smaller doses than those employed by Doctor Lehman, for instance, a considerable series in which 250 units of heparin were employed, no recognizable benefit. They then increased the dose of heparin to 500 units, again with no demonstrable reduction in the amount or extent of adhesions. However, in a third series in which 1,000 units of heparin were employed as an initial dose and two successive similar doses given each succeeding day, so that, in all, the dog received 3,000 units intraperitoneally, they have obtained a beginning promise of result. Now, it will be noted that only in this last series is their dose at all comparable to the amount employed by Doctor Lehman in his very promising work. So that their first statement is, so far as they have gone, these smaller doses are ineffective.

They have reexplored one-half of their current series of cats in which three successive doses of 1,000 units have been employed, and out of a series of eight animals so explored, one showed notable reduction or absence in the amount of adhesions discovered at the second exploration. Now, that is as far as they are prepared to make any statement, and obviously no generaliza-

tions can be made from such fragmentary facts; nevertheless the work is so important, if successful, and so promising in theory, that we expect to go on with it, exploring different modifications, and we hope that Doctor Lehman has at last found a really effective solution of the troublesome problem of adhesions.

Dr. J. Albert Key (St. Louis, Mo.): I would expect that if heparin were present in sufficient amounts to prevent formation of fibrin, there would be some delay and difficulty in the healing of the wound and I would like to know whether that has been noted.

Dr. Edwin P. Lehman (University, Va., closing): The problem of wound hemorrhage following the use of heparin, which Doctor Key has inquired about, was, of course, in our minds. The experience of those who have used heparin extensively in vascular surgery, both in the laboratory and in the human being, has been that there has been no trouble with this complication. With intraperitoneal administration of heparin the animals showed a definite prolongation of the coagulation time, which lasted about 12 to 18 hours. The coagulation time is increased from a normal of three minutes to a maximum of about 12 minutes. In none of them did we have wound hemorrhage, or any disturbance of healing which could be attributed to bleeding.

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BRIEF COMMUNICATION

A DURALUMINUM ENTEROTOME FOR DEVINE COLOSTOMY

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IT IS hardly possible to devise or modify an instrument without incorporating some of the principles of preexisting instruments as well as maintaining the contour and general shape of the instrument to be modified.

The need for modifications depends upon some demonstrable defect found at the time the instrument is used. In applying one of the modified Devine clamps to a colostomy it was found that the central opening of the clamp was too small to surround the "abdominal wall bridge" between the two colostomy openings. This produced some squeezing of the "abdominal wall bridge" which resulted in considerable discomfort to the patient. It was predictable that as the abdominal wall increased in thickness, the pressure on the intercolostomy bridge of the abdominal wall would increase.

FIG. 1A.—Enterotome for Devine colostomy. Closed position.

To overcome this defect it was necessary to increase the total diameter of the central opening of the clamp that encompassed the "abdominal wall bridge." To accomplish this, it was important to use a metal that would be light in weight, of considerable tensile strength, and durable. Duraluminum was found to be such an ideal metal—meeting all the requirements noted above. Therefore, the instrument was made of duraluminum of 0.5 cm. thickness and chromium plated. The instrument, as shown in Figure 1, is a modification and adaptation of the Cook's pile clamp to the requirements of the Devine colostomy clamp.*

The clamp is only 17 cm. in length; therefore, the two handles (Fig. 1, D and C) protrude for only 6 cm. above the surrounding skin surface. The central opening, which encompasses the "abdominal wall bridge" between the two colostomy openings, is 5 cm. in the transverse and 4 cm. in the perpendicular diameters. Therefore, the opening will accommodate the abdominal

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* The instrument is manufactured by V. Mueller and Co. of Chicago, Ill.

wall intercolostomy bridge in either the obese or slender patient without difficulty or discomfort. The blades of the clamp (Fig. 1 A and B) are 5.5 cm. long and have an interlocking, serrated edge on the crushing side of the blade. The two limbs of the clamp interlock and are held together by a lock screw (Fig. 1 E). The blades are pulled together to produce the crushing of the interposed septum by the bolt and wing nut (Fig. 1 D).

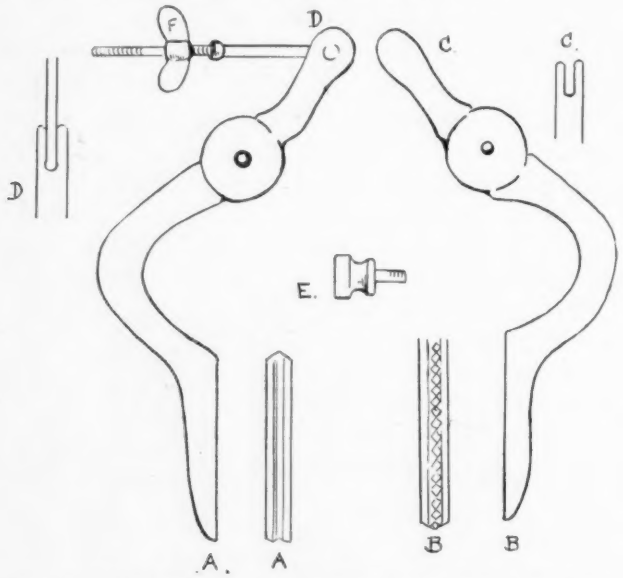

FIG. 1B.—(A) Closed position. (B) Disassembled, showing the component parts.

The application of the instrument to the colostomy is very simple. The blades of the clamp are separated by removing the lock screw (Fig. 1 E) and disengaging the bolt (Fig. 1 F). One blade is inserted into the distal limb of the colostomy. With the forefinger in the proximal limb, the blade is placed in the correct position. The finger is removed and the other blade is introduced into the proximal limb, the two handles are engaged, and the thumb-lock screw (E) is put in place and screwed up tightly. The cross bar (D) is replaced and the wing nut tightened sufficiently to coapt the blades snugly against the colocolon septum. The wing-screw is tightened daily one-half to one full turn until the seventh or eighth day, at which time, by ischemic necrosis, the septum is crushed through. When the spur has been severed, feces exude through the distal colostomy opening.

ERRATA

In the article appearing in ANNALS OF SURGERY, **112**, 240–248, August, 1940, on "Plasma Transfusion in Experimental Intestinal Obstruction" by Doctors Jacob Fine and Samuel Gendel, two errors have occurred: (1) Page 247, the word "for" in the second sentence of the third paragraph, should be "to"; and (2), in the first line of page 248, the figure "208" should be "20.8."